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# STATUS LYMPHATICUS*

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A GREAT deal of nonsense has been written into the subject of Status Lymphaticus, including the recent conclusion of the British Commission that no such condition exists. This conclusion was based upon evidence in the form of bits of tissue from various parts of the body received from pathologists in various parts of the British Empire and accompanied by information on printed cards. It would seem that most pathologists with large experience, particularly in autopsy rooms where sudden death and deaths by crime or suicide are investigated, are convinced that there is a condition, which, for the want of a better name, is called status lymphaticus, which is easily recognizable and which presents definite, and, in many instances, uniform anatomical features. In this so-called status lymphaticus, sudden death sometimes occurs with dramatic rapidity in apparently trivial circumstances and infections are apt to be ill-borne. Perhaps the mere existence of the condition does not always satisfactorily explain the cause of sudden death, but if the anatomical composite were withdrawn from further consideration, as suggested by the British Commission, it would remove at least one important concomitant of sudden death from the field of investigation.

The association of sudden death with enlargement of the thymus was noted by Bichat early in the eighteenth century, but it remained for Paltauf and others of the Vienna school to show that persistence of the thymus in these circumstances is but one of a combination of hereditary anatomical imperfections. Commencing in 1905, Dr. Charles Norris, some time director of laboratories at Bellevue Hospital, made a series of observations on certain peculiarities of configuration in status lymphaticus and called attention to their importance in the clinical detection of the condition. In addition, he pointed out the frequent association of status lymphaticus and certain lesions of the ductless glands, including exophthalmic goiter, Addison's disease, acromegaly and the like, together with its occurrence in suicides and others of unstable emotional qualities.

## DEFINITION

Status lymphaticus may be defined as a combination of hereditary constitutional anomalies, entering into which are certain pecularities of configuration, with preservation or even hyperplasia of the thymus at an age when involution is to be expected, hyperplasia of the lymphoid cells in the spleen, intestine and elsewhere, changes in the distribution of hair, hypoplasia of the vascular system, developmental deficiencies in the genitalia, and,

* From the Pathological Laboratories of Bellevue Hospital.

incidentally, visceral defects of uncertain occurrence and irregular distribution.

Status lymphaticus is compatible with life; many subjects not only attain maturity but survive those involutional changes in the lymphoid tissue which, when completed, deprive the condition of certain of its dangers. It is nevertheless a menace, because it is attended by instability of the lymphoid tissues, providing a mechanism which, when it is set in motion, is capable of so sensitizing the body as to produce anaphylactic phenomena varying in intensity from urticarial rashes to convulsive seizures, asthmatic attacks, or sudden death. The same instability of the lymphoid tissues is apparently responsible for lowering the threshold of infection. It is likewise a menace because it is attended by defective development of the muscular coat of the arteries, sometimes rendering them incapable of withstanding changes in blood pressure which, in ordinary circumstances, are lightly borne.

## CLINICAL ASPECTS

Clinical interest in status lymphaticus seems to be focussed almost exclusively on the fact that life may be terminated abruptly on apparently trivial provocation. As a matter of truth, sudden death in status lymphaticus is a tragedy comparatively rarely enacted, but when it does occur the astonishing rapidity of the blow and the unexpected circumstances in which it is delivered are exceedingly impressive. In addition, status lymphaticus touches certain clinical problems of practical value, which, however, appear to have been almost completely submerged in the contemplation of spectacular death. It will be the purpose of this paper not so much to emphasize the occurrence of sudden death in status lymphaticus as to impress still further the signs by which the condition may be detected in the living patient, the suggestive frequency with which it is associated with acute infections, with certain disturbances of the nervous system and of the ductless glands, with the occurrence of cerebral hemorrhage in the young, and, finally, its clinical significance in lymphoid hyperplasia of the appendix.

In the clinical conglomerate of scrofula the elder Gross long ago recognized the angelic child, in whom he pointed out the existence of a so-called lymphatic constitution and postulated a connection between it and an unusual susceptibility to tuberculosis, particularly of the bones, joints and lymph nodes. It is now known that children of this type are of the category of status lymphaticus.

After puberty the clinical recognition of subjects of status lymphaticus is even more easily accomplished. In addition to the delicate texture of the skin, which may be velvety to the touch, dead white or faintly cream colored, sometimes lusterless or paste-like, but rarely muddy, at other times swept by a faint sheen, and the well-nourished, often muscular, finely proportioned body, with its gracefully outlined arms, rotund and arching thighs and slender waist, it is found that in the male the pubic hairs assume the distribution of the female—that is to say, they are sharply defined in a transverse direction and do not tend to grow toward the umbilicus, while the penis is often small and the glans acorn-shaped. The facial and axillary hairs are scantily developed and the thoracic hairs are few in number or absent. In the female the recognition of status lymphaticus by inspection of the body is rather more difficult, but the diagnosis may be made with relative assurance by the unusually delicate texture of the skin, by accentuation of the graceful outlines of the body and by the presence of small axillary pads with the scanty growth of hair upon them.

## INCIDENCE

In Bellevue Hospital, in a series of 4000 autopsies, status lymphaticus was encountered 249 times, or in 6.2 per cent. The condition occurred in males six times

oftener than in females. Practically every race was represented.

### PATHOLOGY

The anatomical variations in status lymphaticus include of course those external features of configuration and development which have been enumerated, but, in addition, there are changes in other parts that are of importance. These may be classified and described accordingly as they involve (a) the lymphoid tissues, (b) the vascular apparatus, (c) the genitalia and (d) under the heading of incidental anomalies of development.

*Anatomical Types.* In our autopsy protocols at Bellevue Hospital we are accustomed to describe two anatomical types depending upon the condition of the lymphoid tissues: status lymphaticus and recessive status lymphaticus. The former is associated with unusually well-developed changes in the lymphoid tissues and occurs at an age when these tissues are naturally flourishing. The recessive type is marked by atrophic changes in the lymphoid structures, varying in extent according to the time of involution, the configuration remaining the same except insofar as it may be modified by the waste of disease or age. Of the 249 cases there were 118 instances of status lymphaticus, 89 cases of recessive status lymphaticus and 42 cases in which the lymphoid tissues were in places hyperplastic, in other places atrophic, in other words, borderline examples, which, however, were tending in the direction of regression.

### LYMPHOID TISSUES

Of the 118 cases of well-developed status lymphaticus the thymus gland was hyperplastic in every instance. The weight of the gland was recorded in 66 cases. The youngest subject was a child of eight hours whose thymus weighed 70 gm. and in whom death occurred suddenly; the oldest was an acromegalic individual, aged thirty-eight years, in whom the thymus also weighed 70 gm. and in whom death was due to an intercurrent affection. The largest gland to be encountered weighed 115 gm.

The faucial, lingual and pharyngeal tonsils were hyperplastic in about 50 per cent of the cases, and the follicles of the intestinal tract in 88 per cent. The spleen showed no changes worthy of note so far as size was concerned, but was rich in hyperplastic lymphoid follicles (88 per cent). The mesenteric, axillary, inguinal, cervical and peribronchial lymph nodes were practically negligible.

### HISTOLOGY

The histology of the thymus gland in most subjects of well-developed status lymphaticus presents nothing worthy of note other than hyperplasia of the cortical lymphoid follicles, but in many cases this is extreme. In other cases it is to be observed that there are large blood sinuses in the thymus and that they contain greatly increased numbers of lymphocytes. Whether this has any connection with the lymphocytosis which is sometimes encountered in the peripheral blood is a question to which, unfortunately, we are unable to give a definite reply. In Graves' disease, however, it is known that lymphocytosis is a frequent occurrence and that the thymus is hyperplastic often to an extraordinary degree. Shridde and others have pointed out that those patients in which there is a lymphocytosis of 40 per cent or over are dangerous operative risks and are liable to sudden death, which is in keeping with the known occurrence of sudden death in Graves' disease in patients with persistent and hyperplastic thymi. That the lymphocytosis of Graves' disease is due to expulsion of thymic lymphoid cells into the circulation seems reasonably assured in view of the rich numbers of lymphocytes commonly to be found in the blood vessels of the persistent and hyperplastic thymus. No doubt the hyperplastic lymph nodes also contribute their share of lymphocytes. In any event the number of lymphocytes in the circulating blood

may be accepted as a more or less reliable index of the degree of lymphoid hyperplasia and should be taken into consideration in estimating operative risks in subjects of status lymphaticus.

*Necrotic Changes in the Lymph Nodes.* Microscopic examination of the lymph nodes in status lymphaticus shows the presence of increased numbers of lymphocytes, the hyperplasia tending to bring about rearrangement of the architecture of the node by increase in the size and number of the so-called germinal follicles.

In a considerable majority of all cases of well-developed status lymphaticus, but especially often in subjects who have met death suddenly and in response to apparently trivial provocation, the lymph nodes show a peculiar and characteristic change in the shape of cells and the discharge of nuclear dust into the intercellular spaces. In still other cases of status lymphaticus, particularly in subjects who have passed the age of puberty, and in recessive status lymphaticus, the germinal follicles often show almost complete replacement by whorl-like collections of spindle cells, representing, apparently, connective-tissue elements. Lying among the spindle cells are to be made out rather large polyhedral forms resembling degenerate cells of the type of large lymphocytes. The cytoplasm of both the spindle and polyhedral cells stains pink with eosin, so that the follicle stands out in contrast to the deep blue in the small lymphocytes in the immediate vicinity. We have yet to observe them in any condition other than status lymphaticus. They occur not only in follicles of the lymph nodes themselves but in those of the intestine, the lingual, faucial and pharyngeal tonsils, in the follicles of the spleen, and on at least one occasion, I have seen them in the hyperplastic follicles in the interstitial tissues of the thyroid gland of Graves' disease.

The histological changes in the several lymphoid depots in the older cases of status lymphaticus, including, of course, the recessive type, reveal simple diminution in the numbers of lymphoid cells usually with but sometimes without the presence of necrotic, degenerative or fibrotic alterations in the germinal follicles.

## DEATH IN STATUS LYMPHATICUS

The histology and distribution of the lymphoid tissues are, I believe, of fundamental importance in the interpretation of anaphylaxis as a cause of sudden death and of certain convulsive phenomena occurring in subjects of status lymphaticus. Thus, the lymphoid tissues are most abundantly developed in youth and early adolescence, tending to disappear as age advances, which is in keeping with the observation that in status lymphaticus the danger of sudden death is greatest during the period of life in which the lymphoid tissues are most flourishing and is almost unknown in the recessive stage. It is likewise significant that in status lymphaticus those lymphoid tissues which display the highest degree of hyperplasia are to be found in the mucous membrane of the gastrointestinal tract, where, of all the lymphoid structures, they are the ones most actively engaged in the process of filtration, in the course of which they are constantly subjected to the destructive action of a great variety of toxic substances, both chemical and bacterial. In children suddenly dead with all the physical attributes of status lymphaticus, microscopic examination of the lymphoid tissues throughout the body shows, with rare exceptions, myriads of necroses in the germinal follicles of the sort already described. These changes indicate, I believe, that sudden death in status lymphaticus is connected with the release of nucleoproteids formed as a result of destruction of innumerable germinal follicles. It is scarcely conceivable, however, that death is directly traceable to the toxemia produced by sudden and simultaneous destruction of a large number of follicles, since the interval between injury and death is far too short, in the majority of cases, to permit so many necrotic lesions to occur throughout such a

wide distribution. It is more probable, I think, that death is of the nature of an anaphylactic reaction, sensitization being expressed in structural terms by the necrotic germinal follicles, and, chemically, by the release of nucleoproteids which, although not strictly foreign, are none the less pathological, and are comparable, in a toxicological sense, to alien products. After the expiration of the so-called anaphylactic incubation period the lymph nodes are again subjected to the action of destructive substances which serve to bring about still further disintegration of germinal nuclei, thus providing the requisite quantity of specific proteid to complete the anaphylactic cycle. The destructive substances in question may be introduced in the form of antitoxins hypodermically injected, or as vaccines applied by scarification or otherwise, or as substances which have escaped destruction or modification by the hyperplastic lymphoid follicles in the intestinal tract, or which have been manufactured in the process of shock induced even by such simple procedures as the prick of a needle, sudden immersion in cold water and similar events.

That acute necroses may occur in irregular showers is shown by the presence of all stages of both the necrotic and reparative processes in the same lymph node or in different nodes in the same body. This fact is of moment in that the anaphylactic reactivity of the body is determined by the number of acute necrotic lesions in the germinal follicles, or, in other words, the anaphylactic incubation period in man, as in experimental animals, varies with the initial dose of the sensitizing proteid. For example, in guinea-pigs injected with small amounts of horse serum from twelve to fourteen days suffice for sensitization. With larger doses, however, the interval may be extended to weeks or months. In subjects of status lymphaticus it is naturally to be expected that early sensitization follows a small shower of necroses and that larger showers are succeeded by a longer period of incubation, the reactive ranges thus varying within wide limits, both in the matter of time and intensity. At one moment the tissues are exquisitely tuned and await only the receipt of a sufficient quantity of specific protein to react violently, even to the extent of sudden death, while at another moment events are so timed that the same quantity of specific protein exerts no such effect, a fact which tends to explain why certain subjects of status lymphaticus survive surgical and other procedures which, in others, are attended by disaster. In this instance it is apparently a question of anaphylactic reactivity dependent upon the number of acute necrotic lesions in the lymph nodes and upon the interval which has elapsed since their inception.

### STATUS LYMPHATICUS AND CEREBRAL HEMORRHAGE IN THE YOUNG

In status lymphaticus the cerebral vessels, particularly the arteries entering into the formation of the circle of Willis and the branches which emerge therefrom, are often small in caliber and extremely thin and delicate. As seen at autopsy the larger vessels at the base, for example, are collapsed, one wall lying against the other and, when removed from the body and viewed by transmitted light, are easily translucent. Upon microscopic examination these vessels show simple hypoplastic alterations, the deficiency being most marked in the muscular coat, which is distinctly less developed than in normal circumstances. The exact incidence of these changes in status lymphaticus has not been determined but the association has been noted with such frequency as to leave no doubt that the two are companion manifestations of one and the same underlying developmental defect.

Hypoplasia of the cerebral vessels in subjects of status lymphaticus is of profound importance from the standpoint of sudden death, not only sudden death ensuing spontaneously, but in subjects who have been exposed to a degree of injury

which, in normal individuals, would be regarded as insignificant. In the latter group of cases its medicolegal application cannot be overestimated. Cerebral hemorrhage in young, non-syphilitic subjects of status lymphaticus occurs a number of times in the course of every year, and is due to rupture of hypoplastic cerebral arteries occurring spontaneously or upon the receipt of apparently trivial traumatism or under the influence of physical strain or intense excitement. In such subjects, therefore, any influence which causes sudden increase in intracranial blood-pressure may be disastrous, such as heavy lifting, straining at stool, the tension of argument, fighting, etc.

Hypoplasia of the smaller blood vessels in status lymphaticus is apparently an element of danger in workers in compressed air, 5 cases of caisson disease having been examined postmortem in Bellevue Hospital in individuals showing the characteristic stigmata. All of them were young, muscular adults. All of the bodies presented innumerable odorless air bubbles in the vessels of the brain and trunk. From these 5 cases, which represent the sum total of our experience in caisson disease during a period when active tunnelling operations were under way in New York City, it is quite apparent that status lymphaticus constitutes a contraindication to the acceptance of a workman for duty in an atmosphere of compressed air even though decompression be brought about with scientific exactitude. Just why this should be true is not apparent, but it probably has something to do with defective permeability of congenitally hypoplastic blood vessels, so that tissues saturated with nitrogen under pressure yield this gas under decompression in such quantities that the blood cannot take it into solution and it circulates as emboli.

Sudden death in subjects of status lymphaticus has often been ascribed to pressure of the enlarged thymus upon the trachea. At Bellevue Hospital we have not been able to implicate the thymus as a mechanical factor in the production of death, the anatomical signs of tracheal compression and of death from suffocation never having been found, although sought. In this connection it is significant that the heaviest thymus in the Bellevue cases, namely a gland of 115 gm., was greatly below the weight necessary for compressing the trachea, as shown by experiment, which according to Tamassia is at least 180 gm.

### STATUS LYMPHATICUS AND EMOTIONAL INSTABILITY

Emerson, working in the alcoholic wards at Bellevue Hospital, examined 1000 patients suffering from the effects of attempted suicide and from cocaine, morphine, heroin, alcohol and similar habituating drugs. Of this number 22 per cent showed most or all of the physical stigmata of status lymphaticus. In the autopsy rooms at Bellevue Hospital, which are utilized for the performance of autopsies in medicolegal cases, it is a matter of almost daily observation that suicides and degenerates, and gunmen and other criminals who have met death by violence, present the characteristic conformation and anatomical changes of status lymphaticus. According to the combined statistics of Bartels and Miloslavich, amounting to 232 cases of suicide, 80.5 per cent showed the signs of status lymphaticus at autopsy. Among epileptics and the insane the condition is common (Karpas).

### STATUS LYMPHATICUS AND EXOPHTHALMIC GOITER

The association of status lymphaticus and exophthalmic goiter, while not constant, is common, and is particularly noteworthy in connection with the incidence of status lymphaticus among the emotionally unstable. Capelle collected 60 cases of Graves' disease from the literature. In 44 per cent the patients died from intercurrent affections, and in all of them the thymus was persistent and hyperplastic. Of those dying of Graves' disease without

associated lesions, 82 per cent presented enlargement of the thymus, while in cases of sudden death following operation for removal of the thyroid the thymus was hyperplastic in all.

It has long been known that derangement of the emotional nervous system, manifested by intense grief or fear, may be rapidly followed by the symptoms of exophthalmic goiter, that exophthalmic goiter frequently is implanted in young women of uncertain emotional balance, sometimes in a family whose genealogy is marred by a history of epilepsy, insanity and kindred disorders, and that a certain proportion of all cases of Graves' disease follow influenza or streptococcal throat infection, either of which is prone to leave its victim mentally perturbed. That Graves' disease not infrequently follows shock was exemplified by the World War, numbers of soldiers exposed to the terrifying vicissitudes of trench life having been invalided with symptoms of Graves' disease varying in intensity from simple tachycardia and muscular tremors to the fully developed picture. It seems not improbable that exophthalmic goiter, in certain subjects of status lymphaticus at least, is the result of disturbances in an already unstable nervous system that have so altered the function of the thyroid gland as permanently to externalize the physical attributes of fear as expressed by protruding eyeballs, irregularity and increased rapidity of the heart's action, muscular tremors, clammy sweats, pallor and the like. In other words, the subject of status lymphaticus, as a result of hereditary anatomical imperfections, appears to be more susceptible to those influences, infective, psychic or otherwise, which precipitate exophthalmic goiter than the individual who more nearly approaches the normal. This hypothesis, at all events, is more in consonance with established facts than the extreme view of certain students of the French school who maintain that Graves' disease is the direct result of disturbances in the thymus gland rather than the thyroid, and who have advocated and actually practiced thymectomy for its relief. The alternative, as far as I can judge, is to dismiss the association of status lymphaticus and Graves' disease as accidental, and this seems scarcely justified by circumstances.

### CHLOROSIS

It was pointed out by Virchow that in chlorotic girls the aorta is hypoplastic and the genitalia, notably the uterus, of the infantile type. Not only are these deficiencies common to status lymphaticus in the female, but it is a matter of observation that chlorotic girls invariably present a skin of exquisite texture and that the normally graceful outlines of the body are accentuated and the subcutaneous fat well preserved. That the vasomotor system is easily disturbed is shown by frequent changes in color, by the paradoxical association of rosy cheeks with pronounced anemia, the so-called chlorosis rubra, by dermatographia and by a tendency to syncope which is often out of proportion to the severity of the anemia, while emotional disturbances are notoriously common. In short, the anatomical signs and the symptoms of chlorosis suggest that this form of anemia is an incidental and fugitive condition in girls with status lymphaticus.

### STATUS LYMPHATICUS AND ACUTE INFECTION

There is little in the literature bearing on the susceptibility of subjects of status lymphaticus to infection. Daut has shown, however, that over 25 per cent of patients dying of diphtheria present the anatomical changes of status lymphaticus. Elser's observations on epidemic cerebrospinal meningitis lead him to the conclusion that status lymphaticus, occurring in over a quarter of all subjects examined at autopsy, constitutes a predisposing factor in this variety of infection, and, in addition, he noted that the issue is apt to be rapidly fatal in patients of this sort. In 1906–1907

there was an epidemic of cerebrospinal meningitis in New York City and 55 cases were submitted to autopsy at Bellevue Hospital. Of this number 33 (60 per cent) occurred in young subjects of status lymphaticus. Of the 33 cases it was possible to ascertain the duration of symptoms in 30. Death occurred in 70 per cent after the lapse of an average interval of 3.4 days.

### LYMPHOID HYPERPLASIA OF THE APPENDIX

In the routine examination of appendices, I have long been impressed by the fact, that, of all appendices removed at operation from subjects of status lymphaticus, a considerable number reveal no noteworthy microscopic alteration other than lymphoid hyperplasia, with or without the association of sclerotic changes in the connective tissues. Signs of inflammatory exudation are missing, and yet the patients are subjected to operation because of disturbances consisting of one or more attacks referable to the region of the appendix characterized by cramp-like pain, or pain and moderate tenderness lasting for several hours or days. Muscular rigidity, if present at all, is rarely of marked intensity, nausea is not uncommon but vomiting is rare. The temperature is usually normal, the pulse rarely exceeds 80 or 90 beats, and headache and constipation are fairly common. The leucocytes are within normal limits, the differential count disclosing no noteworthy variations in the number of polymorphonuclear cells.

Microscopic examination reveals changes characterized by hyperplasia of the germinal follicles, and, after the occurrence of several attacks, by sclerosis and finally by obliteration of the appendix.

### SUMMARY

1. There is a condition which, perhaps, for the want of a better term, is known as status lymphaticus.

2. Status lymphaticus may be defined as a combination of hereditary constitutional anomalies, entering into which, among other things, are certain peculiarities of configuration, with preservation or even hyperplasia of the thymus at an age when involution is to be expected, hyperplasia of the lymphoid cells in the spleen, intestines and elsewhere, changes in the distribution of hair and hypoplasia of the vascular system.

3. Status lymphaticus is compatible with prolonged life, but occasionally it is associated with sudden death on trivial provocation due, apparently, to anaphylaxis, or to spontaneous rupture following increased pressure acting on a congenitally hypoplastic cerebral vessel.

4. Status lymphaticus is encountered in about 6.2 per cent of all autopsies. It occurs in males six times oftener than in females.

# PULMONARY EMBOLISM FOLLOWING SURGICAL OPERATION

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FATAL pulmonary embolism is a major tragedy, particularly when it afflicts patients following surgical operation, for almost all such patients are well on the road to recovery and without this catastrophe would probably be completely restored to a normal state of health.

The clinical histories of these patients are chiefly remarkable because of sameness. The age is thirty years or older. The weight is likely to be more than average. The operation is abdominal, and the more serene the events during and following this operation, the more likelihood there is of venous thrombosis and resulting embolism. There are no major complications such as peritonitis, pneumonia, or formation of abscess. Convalescence is peculiarly uneventful. Suddenly, almost always out of a clear sky, there is strange restlessness, rapidly ensuing shock with substernal distress, air hunger, and collapse and death usually in two to fifteen minutes. The horror of the patient's relatives is echoed by the poignant distress and shock of the attending surgeon, colleagues and nurses. After surviving the ordinary perils which may accompany any operation, and after assurances of cure are clearly manifest to all, like a stroke of lightning, this unexpected agent of the grim reaper appears on the scene and wipes out at one blow the results of the otherwise successful operation and postoperative care.

Examination after death reveals the pulmonary arteries plugged by a coiled up and folded antemortem clot which is stuffed into each main artery a short distance from the bifurcation, and which projects backward toward the pulmonary valve and the right auricle. If the arteries are completely closed, death has been sudden. If even a slight cleft remains open life may have been prolonged minutes and even hours. On further search, the remains of this clot usually can be discovered in the branches of the left iliac vein, either the internal iliac or the femoral. The wound itself, as well as the peritoneum and lungs, is usually comparatively free from evidences of infection.

The clinical course, the cause of death, and the pathologic picture of pulmonary embolism all seem to be simple, and perhaps by their very simplicity have defied all efforts to foresee clearly the occurrence of the condition, prevent its happening, or explain its cause.

Quite evidently the blood has clotted in the smaller veins in life, and this clot has propagated itself rapidly toward and into the larger veins (Fig. 1). Suddenly some slight movement of the body has broken off a large piece, and this has floated in the blood of the vena cava, through the right side of the heart, and into the pulmonary artery and its branches (Fig. 2).

If the propagating process has been slow enough, the clot adheres to the wall of the vein and the leg becomes swollen and painful, but the danger of the clot loosening or breaking off becomes materially lessened. In short, we do not fear so much the manifest presence of venous thrombosis as we fear its too rapid extension. This is easily proved by the fact that thrombosis is a distinctively conservative event. Without its action no operation could be successful, life itself could not continue. We *expect* the cut and crushed ends of blood vessels to be plugged by thrombi. But we do

not expect that this life-saving process will continue on into vessels where the blood is in motion, and when it does, we are in

Fig. 1. Thrombosis of left internal iliac vein with extension to vena cava.

the presence of what might be designated as pathologic thrombosis. The difference then, between the conservative or physiologic, and the pathologic, phases is one not of kind but of degree. Formation of thrombus becomes abnormally active, and is extended even into the stream of circulating blood.

Such pathologic occurrences are comparatively common in the body. The saccular aneurysm is usually more or less filled by a thrombus. The infarcted and dilated left cardiac ventricle has a thrombus plastered to its comparatively immobile wall. In cardiac decompensation from any cause, the auricular appendages, and then the auricles, become the seat of thrombosis.

Even more frequent is the occurrence of pathologic thrombosis in the prostatic plexus of veins. Almost every adult male, past middle life, harbors phleboliths in this plexus as the result of previous formation of thrombi and at death from any

Fig. 2. Massive embolus plugging pulmonary artery.

cause this plexus is likely to be distended and filled with antemortem clots. The cervical and vaginal plexuses in the female are similarly, but less frequently and less extensively, affected.

No exhaustive analysis is necessary to determine the principal factor which determines the pathologic thrombus in each of the foregoing examples. It is surely neither injury to the vascular wall, nor infection, nor increase in the coagulability of the blood. It is simply stasis or stagnation of the circulating blood at these particular situations. This stasis may occur because local dilatation and consequent loss of motility of some portion of the vascular system has taken place, or because there is actual decrease in the force or

rapidity of the heart's action, thus slowing the entire blood-current.

It is this latter condition with which we have to deal in cases of pulmonary embolism following surgical operations on the abdomen.* In the first place the patient is forced to lie quietly in bed over a considerable period of time. The pulse rate and blood pressure tend to become lower. Then not only the abdominal wall, but the whole body is rather effectually "splinted" by the operative wound. Respiratory movements are checked, abdominal breathing and all of the other activities connected with muscular contractions of the walls are inhibited, and movements of the entire torso are markedly reduced, especially in the first week following the surgical wound. The maximal effect of these reductions is seen following this first week, and it is in the second week that the largest number of fatal embolisms is seen.

On the contrary, if infections develop, giving a rapid pulse and increased respiratory rate, this accident rarely happens. Operations on other portions of the body do not tend to limit the activity of the circulation of the pelvis and lower extremities, and it is in these situations that thrombosis becomes most dangerous. It is true that thrombi form following these other operations, and occasionally pieces break off and go to the lungs, but the vascular trees of these operative fields are not so favorable for their pathologic formation or, what is more important, their abnormal propagation.

Operations on patients affected with thyrotoxicosis are almost never complicated by embolism, the rapid heart again serving as a factor of safety.

As stated, the veins of the pelvis and the legs, usually the left,* are the common sites of thrombosis which may produce fatal embolism. The propagation often

Fig. 3. Fatal pulmonary embolism.

starts in the veins of the pelvis. This is particularly true when operations are in this region. Proceeding up the internal iliac vein, the external iliac vein is occluded, and the stage is set for the freeing of a large clot in the venous blood. Varicose veins of the legs may also serve as primary foci. So far as this lesion is concerned these veins constitute a place of lessened resistance. Of course, in addition to the immediately fatal type of pulmonary embolism (Fig. 3), smaller emboli are frequently released and occlude branches of the pulmonary artery (Fig. 4), with subsequent production of pulmonary infarcts. These may eventually, by infection, bring about death. Probably, much more frequently than is realized, smaller emboli are swept into the lungs without causing any appreciable clinical or pathologic phenomena.

The menace of thrombosis and embolism is then ever present, and the question of its prevention should be a very live one. If, as has been asserted, some hidden infection is the cause, there is little hope of success, as is also true if the processes could be shown to be due to increased

* The factors mentioned previously may, and undoubtedly do, play minor parts, and occasionally each of them may assume a major position in certain well-known conditions. Thus, the roughened wall of the aorta promotes thrombosis, as does the infected heart valve or the suppurative foci in the regional vessels, and abnormal increase in the power of coagulation of the blood may promote intense and even fatal intravital clotting.

* Probably because of the pressure of the right common iliac artery at its crossing over the left common iliac vein.

coagulability of the blood. Any attempt to lower the power of coagulation of the blood is likely to prove as dangerous as the condition we are endeavoring to combat. If, however, as seems likely, stasis in the venous channels is the underlying cause, there is every reason to attempt prophylactic procedures which are designed to increase the activity of the blood flowing in these regions of stagnation, for it is quite clear that the more rapidly the blood flows, the less tendency there is to the growth of intravital thrombi.

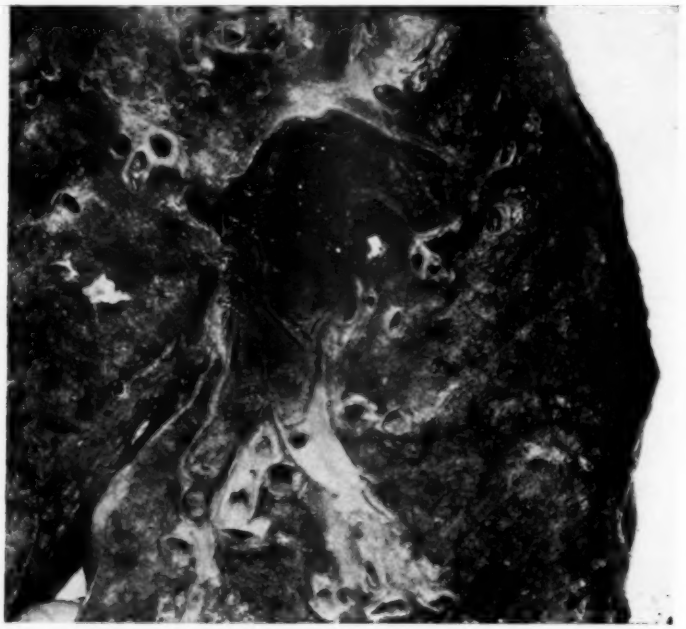

Fig. 4. Complete blocking of pulmonary artery by embolus.

It is not within the province of this paper to advocate any particular measures for bringing about this increased flow in the veins of the pelvis and lower limbs. It is sufficient, perhaps, to point out that massage at frequent intervals, passive and active motion of the legs, frequent moving and turning of the body, and encouragement of an increased respiratory rate, such as deep breathing, are logical procedures. Even more important is the local use of heat. Keeping the feet and legs warm, and even applying heat locally to the pelvis, might prove deciding factors. The use of vascular stimulants, such as thyroid extract, might be indicated in selected cases, but caution in their general employment is clearly necessary. Even when so-called thrombophlebitis is recognized as being present, all of these methods, except massage and motion, can still be used. The important thing is to recognize the possibility, and even probability, of the development of this lesion, and to endeavor to find some practical method for actively combating its inception.

# THROMBOSIS OF THE VEINS OF THE LOWER EXTREMITY AND PULMONARY EMBOLISM AS A COMPLICATION OF TRAUMA

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OCCASIONALLY a thrombus forms in one of the veins of the lower extremity and becomes an embolus which occludes the pulmonary artery. When this occurs death follows immediately from acute asphyxia. A great variety of medical and surgical patients die as a result of this complication. It is noted especially after abdominal operations, in the course of pregnancy, in many medical conditions, and as a sequel to different types of injury. The last mentioned group forms a small but distinct proportion of the causes of death in any large series of traumatic cases.

It is my intention here to discuss the incidence of pulmonary embolism in the traumatic cases collected from the Office of the Chief Medical Examiner of New York City and to discover, if possible, what factors tend to produce this complication.

It is advisable at the start to give a brief account of the process of thrombosis and the way in which the thrombus becomes an embolus. First, thrombosis can be defined as the formation of a clot in the blood vessel during life from the constituents of the blood. As described by MacCallum[1] the process starts as a gray translucent collection of blood platelets on the vessel wall, known as the head. On this as a foundation other platelets are deposited by the flowing blood to form rows of laminae which run transversely to the long axis of the vessel and project into the lumen. These laminae anastomose freely with one another and form a framework to which adhere numbers of leucocytes from the circulating blood. A thromboplastic substance is produced and fibrin is formed between the laminae. In the fibrin meshwork large numbers of red blood cells are imprisoned which give the thrombus a predominantly red color. In the early stages of the process the laminae may project slightly above the surface of the growth as grayish corrugations known as the lines of Zahn.

When the thrombus has grown large enough to occlude the vessel, stasis occurs and the blood collects around the proximal and distal ends of the obstruction, depending upon the distribution of the venous tributaries in these regions. The blood in either event stagnates from the thrombus to the inflow of the next branch. This column clots en masse, like blood clots in a test tube, is dark red in color and may resemble a postmortem clot quite closely. This part of the thrombus is attached to the head and is known as the tail. The growth of the tail may stop at the nearest point of inflow or the process may begin again with further platelet deposits. It may extend along the course of the vessel in a distal and in a proximal direction, continually growing by accretions to either end of the thrombus. In a vessel like the femoral vein it may become as long as 15 to 25 cm. and it is usually as thick as the femoral vein itself, which is about 10 to 15 mm. in diameter.

In some cases the thrombus in the femoral vein may free itself from its attachments and travel through the inferior vena cava and right side of the heart to blockade the main branch of the

[1] MacCallum, W. G. A Textbook of Pathology. Ed. 3, Phila., Saunders, 1926.

pulmonary artery. This event happens in various ways. Occasionally the entire thrombus, if it is coherent enough, may be transported en masse like a small piece of rope (Fig. 1) and, because of the twists and turns in the right heart, may be bent, coiled or even knotted when it reaches the pulmonary artery. In other cases the clot may not be as coherent and, in its journey through the right heart, it may be broken into a number of small pieces about 2 to 5 cm. in length (Fig. 2). In either event the embolus impedes the flow of blood through the lungs and causes death suddenly and without warning.

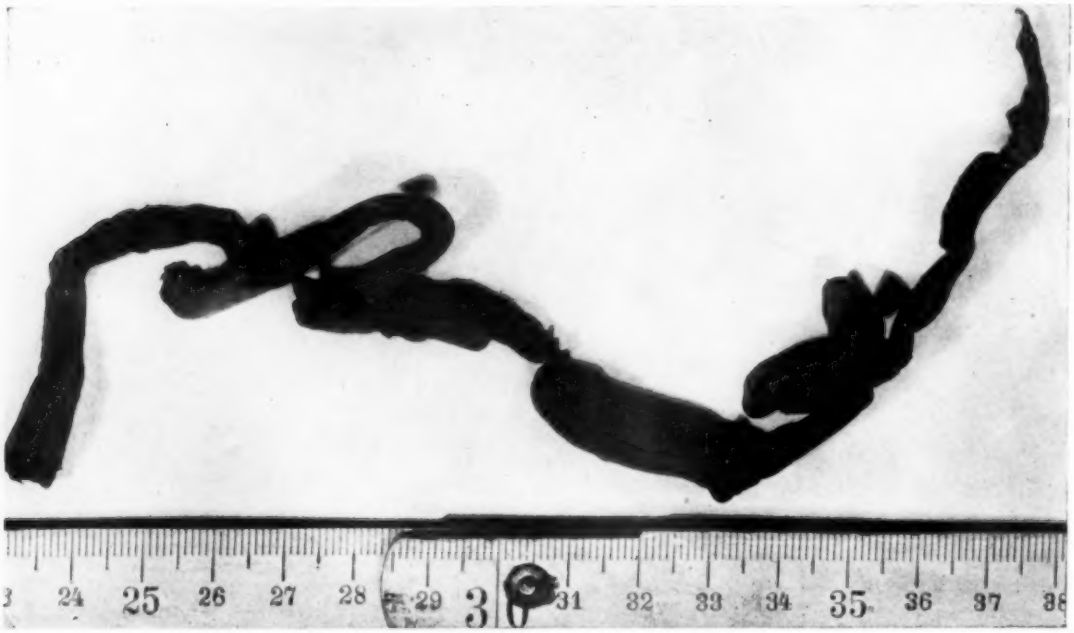


Fig. 1. Long coherent pulmonary embolus.

In some cases the thrombus formed in the femoral vein is not coherent at all and small pieces may detach themselves from the vein to form an embolus to a small branch of the pulmonary artery in the lung tissue. These small emboli cause small hemorrhagic infarcts in the lung tissue which may produce symptoms of pain in the chest, expectoration of blood, etc. Premonitory signs thus are given that a clot is forming in the femoral vein so that the death a few days later by a large embolus is not unexpected.

In the vast majority of cases the embolus in pulmonary embolism is composed of bland, non-septic thrombotic material. Under the microscope, a section of the embolus stained with hematoxylin and eosin shows a homogeneous pink framework of blood platelets with fibrin, red blood cells and a few leucocytes in between. The platelet framework is characteristic and if it can be demonstrated satisfactorily is proof enough that the structure is a real thrombus.

The different factors which contribute to the development of thrombosis and pulmonary embolism have been discussed at great length in the literature. McCartney[2] gives an excellent summary of the subject in his article and lists three which are considered the most important. They are: (1) Injury to the vascular endothelium which may occur from actual trauma, infectious processes and toxic action in general. (2) Slowing of the blood stream which may occur from a general circulatory disturbance or a local condition like a phlebosclerosis, pressure on a vein,

[2] McCartney, J. S. Jr. *Arch. Path.*, 3: 921 (June) 1927.

etc. (3) Changes in the composition of the blood induced by toxic or infectious conditions which renders the blood more liable to intravital clotting. The injury to the endothelial lining of the blood vessel generally is considered the most important factor as it forms the nidus for the head of the thrombus. The slowing of the blood stream and the changes in the composition of the blood are important contributory influences and aid in the future growth of the thrombus.

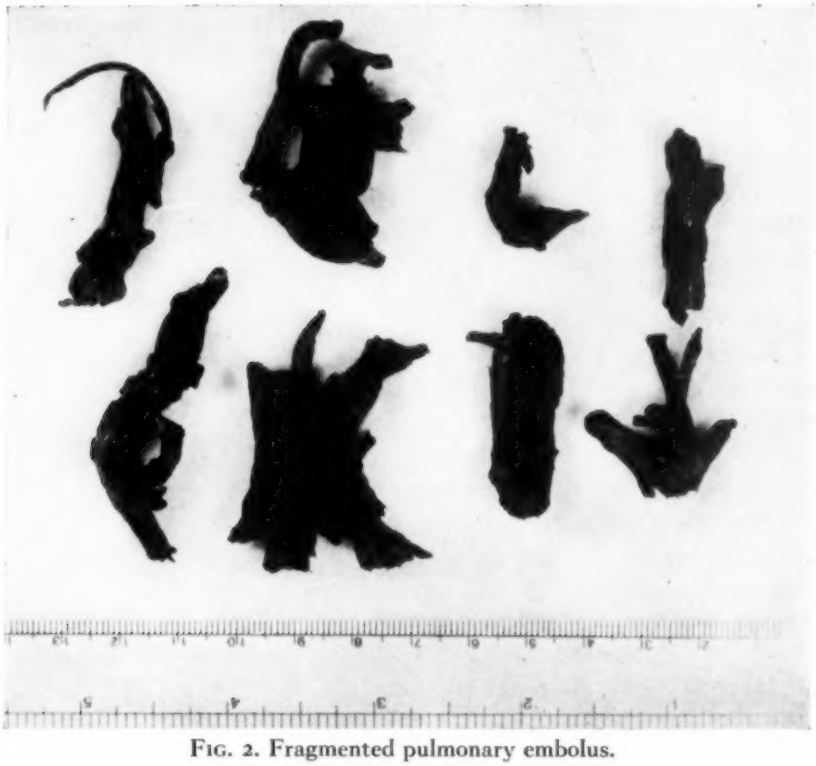

Fig. 2. Fragmented pulmonary embolus.

Pulmonary embolism as a cause of death has received much attention in recent years. Rosenthal[3] in his review has pointed out that there has been a steady increase in the incidence of pulmonary embolism in the clinics of Central Europe from the years 1919 to 1928; after 1928 there was a slight decrease. He attributes this phenomenon to the period of want and social instability which prevailed in these countries during the early nineteen twenties and especially to the rise in the proportion of cardiac and vascular diseases during that period; he considered suppurative and infectious diseases of less importance in the causation of thrombosis. As an added confirmatory support of his point he mentions the fact that in Canada and the United States, where such social conditions did not prevail, there was not any increase in the incidence of pulmonary embolism during the same period.

[3] Rosenthal, S. R. *Arch. Path.*, 14: 215 (August) 1932.

## DESCRIPTION OF THE MATERIAL

The material which I present here comprises 90 cases of thrombosis of the lower extremity with death from pulmonary embolism due to all causes. All of these cases are routine necropsies of the Office of the Chief Medical Examiner of New York City which were performed because of an obvious association of the death with a definite injury or because the deceased has died under suspicious or unusual circumstances. The majority of cases were collected between January 1, 1924 and January 1, 1934, but a small number were added which had occurred in other years.

This material could be divided into a number of different types on the basis of the probable cause of venous thrombosis and pulmonary embolism. They are listed as follows:

| | |
|---|---|
| As a complication of a definite injury | 60 |
| As a complication of a phlebitis with a traumatic history but without a definite injury | 6 |
| As a complication of pregnancy | 3 |
| As a complication of an operation | 4 |
| As a complication of varicose veins | 7 |
| As a complication of other natural conditions | 10 |
| Total | 90 |

The reason for the preponderance of injury cases is because the Office of the Chief Medical Examiner is concerned mostly with deaths which result from all sorts of violence. The other types mentioned would be included in our material at infrequent intervals and more or less fortuitously. Hence only the figures in connection with cases of trauma would have any significance in so far as statistics are concerned.

Among the cases of pulmonary embolism associated with definite injury were 55 which occurred during the ten years between 1924 and 1934. When these were listed in the year in which they died the following results were obtained:

| *Year* | *Cases* |
|---|---|
| 1924 | 2 |
| 1925 | 6 |
| 1926 | 2 |
| 1927 | 4 |
| 1928 | 2 |
| 1929 | 4 |
| 1930 | 10 |
| 1931 | 9 |
| 1932 | 8 |
| 1933 | 8 |

In the years preceding the depression, the yearly average was 3 to 4 cases, but after the depression it was over twice as much. An attempt is not made here to draw any conclusions from so small a series of cases, but it is suggestive that the cases of pulmonary embolism should increase during the years of depression, especially when viewed in the light of Rosenthal's remark concerning the increase in the incidence of this condition in Central Europe from 1919 to 1928.

A detailed examination of the 60 post-traumatic cases of pulmonary embolism disclosed the following facts:

The race incidence was not significant, as 55 were white and 5 were negroes.

The sex ratio was: females 37, males 23. In any large series of cases of pulmonary embolism, the females outnumber the males.

The age incidence only confirmed the prevalent conception that pulmonary embolism is a condition which attacks middle age for the most part. Taken by decades, the 60 cases were grouped as follows:

| *Age in Years* | *Cases* |
|---|---|
| 30 to 39 | 4 |
| 40 to 49 | 10 |
| 50 to 59 | 22 |
| 60 to 69 | 15 |
| 70 to 79 | 8 |
| Over 80 | 1 |

The types of casualty which produced the trauma were as follows:

| | *Cases* |
|---|---|
| Automobile injury | 36 |
| Falls | 16 |
| Hit by falling object | 2 |
| Blow from revolving door | 1 |
| Blow during altercation | 1 |
| Homicidal incised wounds | 1 |
| Bullet wound | 1 |
| Unknown injury | 2 |

The injuries varied from the serious to the slight; they were always definite enough to persist until the patient died of the pulmonary embolus, but were not severe enough to cause death before the embolus had time to develop.

The lesions produced by the trauma were as follows:

| | *Cases* |
|---|---|
| Fractures of femur | 14 |
| Fractures of tibia and fibula | 11 |
| Fractures of tibia | 2 |
| Fractures of fibula | 7 |
| Fractures of pelvis | 5 |
| Miscellaneous injuries of lower extremities | 11 |
| Injuries elsewhere in body | 10 |
| Total | 60 |

The 14 fractures of the femur were injuries of varying grades of severity.

Ten were intracapsular fractures of the neck, 2 were intertrochanteric fractures, and 2 were compound fractures of the shaft at the junction of the middle and lower third. Seven of these fractures were present in the right femur; in 6 of the cases a thrombus was present in the right femoral vein or one of its tributaries, while in one case a thrombus could not be found. The remaining fractures involved the left femur; in 5 of these cases the left femoral vein was thrombosed, and in 2 cases no thrombus was present.

The 11 fractures of the tibia and fibula combined showed simple fractures present in 8 cases, and in 3 cases compound fractures with a slight infection around the broken bones. A simple fracture of the tibia was present in 2 cases and simple fractures of the fibula were present in 7 cases. In this list of 20 cases, the right leg was involved 4 times; in 3 cases there was a thrombus of one of the veins of the right lower extremity, and in one case a thrombus could not be found. The bones of the left leg were fractured in 16 instances; in 11 cases a thrombus was found in one of the veins of the left lower extremity, while in 5 cases a thrombus could not be located.

Fractures of the pelvis were present in 5 cases. In one there was an acetabular fracture on the left side and a thrombus was present in the left internal iliac vein. In 2 of the other cases a left femoral thrombosis was found, but it was not located on the side of the injury. In the remaining 2 cases a thrombus was not found.

Eleven cases of miscellaneous injuries of the lower extremities were described which were listed as follows:

| | *Cases* |
|---|---|
| Bullet wound of the right knee | 1 |
| Traumatic arthritis of right knee | 1 |
| Sprained ankle | 1 |
| Lacerated wound of leg | 3 |
| Contusions and abrasions of legs | 5 |

A right femoral vein thrombosis was found in 6 cases corresponding to injuries on the right lower extremity, while in 5 cases a thrombus could not be located.

Injuries elsewhere in the body were recorded in 10 cases. They were listed as follows:

| | *Cases* |
|---|---|
| Fracture of skull | 2 |
| Fracture of skull and ribs | 1 |
| Fracture of lumbar spine | 1 |
| Injuries of chest | 2 |
| Fractures of ribs | 1 |
| Injuries of head | 1 |
| Fracture of right humerus | 1 |
| Incised wounds of chest and abdomen | 1 |

The left femoral vein was thrombosed in 3 cases, the right femoral vein in 4 cases, and the inferior vena cava in one case. In 2 cases a thrombus could not be located in any of the veins. In this group the site of the thrombus was not related to the site of the injury.

These facts can be summarized by noting that in cases of trauma to the bones and soft parts of one of the lower extremities, a thrombus was found in the veins of that extremity in 32 instances. Of these 32 cases, the right leg was involved 16 times and the left leg 16 times. In 14 injuries of the lower extremities, a thrombus could not be located. Injuries which did not involve one of the lower extremities were described in 10 cases; the thromboses which were found in the veins of the lower extremities did not correspond with the site of the traumatic lesion.

The length of time which elapsed between the onset of the trauma and death from pulmonary embolism was quite variable. The minimum interval was four days and the maximum interval was fifty-four days. The majority of cases showed an average which varied between ten and twenty-four days. The significance of this fact is not clear and can be discussed more advantageously later.

The clinical, anatomic and microscopic features of this condition were studied in 21 cases taken from the larger group of 60. On these 21 cases I had performed the necropsy and had done a microscopic

examination on the femoral vessels and the other organs.

The clinical histories tended to fall into different patterns. In all, the patient had been in some type of accident and had been incapacitated for a shorter or longer time by the injuries received. At the time of their death the patients had recovered from the immediate effects of the trauma or their condition was improving. Sixteen of them died suddenly in the hospital ward, 9 were still confined to bed on account of the injury, while 7 were convalescing and were up and around the ward. Only one of these cases is mentioned as having given premonitory signs; about twenty days after the injury this patient suddenly developed a friction rub and a pain in the right chest which, at necropsy, was found to be due to a recent pulmonary infarct caused by a small embolus. Five of the patients had received injuries which had not incapacitated them unduly and they died suddenly at their residences.

The individuals in this series varied from early middle age to senility and all types of physical conformation were represented, from the poorly nourished to the fleshy type tending towards obesity.

In 12 of the 21 cases the embolus in the pulmonary artery consisted of small clots measuring about 9 to 15 mm. in diameter and 2 to 5 cm. in length. In other instances the emboli were longer and fewer in number. Some were either bent or coiled or knotted, and often had branches corresponding to the tributaries of the femoral vein. Microscopic examination of some of these clots usually disclosed the characteristic platelet framework. In 5 instances recent hemorrhagic infarcts were found in the lungs, indicating that small emboli had preceded the large fatal one.

Associated morbid conditions of greater or lesser severity were found in 17 of the 21 cases. They were:

| | *Cases* |
|---|---|
| Generalized arteriosclerosis | 9 |
| Luetic meningitis and aortitis | 2 |
| Thrombosis of a cerebral artery and cerebral infarct | 1 |
| Carcinoma of uterus | 1 |
| Uterine polyp | 1 |
| Purulent pericarditis | 1 |
| Subacute infection at point of injury | 2 |

The veins of the lower extremities, however, showed the most significant lesions. Only 5 cases of the 21 disclosed varicose veins on external examination. In the remaining 16 there was no external evidence that the venous circulation was abnormal in any way, even when the main femoral vein was occluded by a thrombus. Microscopic examination generally disclosed, however, that some type of inflammation was present in the vein wall or in the tissues immediately adjacent.

In those cases in which thrombosis was found, the thrombus was confined to the veins of one extremity. The veins of the other leg were not thrombosed and did not show any inflammation on section, though the vein wall might show a slight phlebosclerosis.

A closer consideration of the veins which were thrombosed showed the thrombus as a red or dark reddish brown clot distending the vein or simply attached to the wall as a small mural growth. The larger veins in about 80 per cent of the cases showed a definite phlebosclerosis, that is, there was fibrous thickening of the wall and fibrous plaques on the intima. In most cases there was an inflammatory infiltration of lymphocytes and in a few instances of plasma cells and of eosinophiles in the vein wall or in the perivascular tissues. This infiltration varied from small cellular collections around the vasa vasorum to marked diffuse infiltration of the wall of the vein. The thrombus was frequently included in the section and in many instances the platelet laminae were demonstrated. In a few cases a process of organization was present which started from the intima and invaded the margin of the thrombus.

The following case, which was taken from the small series of 21 cases, illustrates quite well some of the pathologic lesions in the veins.

A white woman, thirty-seven years of age, was struck by an automobile about three weeks prior to death. Her right knee was injured, but aside from a moderately severe traumatic arthritis she suffered no ill effects from the accident. Two weeks later she began to bleed freely from the uterus and soon suffered from loss of blood. She was admitted to a hospital for her uterine condition. On the fourth day of her stay she died suddenly in an attack of acute asphyxia. As far as could be ascertained, the patient had been perfectly healthy prior to the accident.

The necropsy disclosed a woman of about the age stated, of good muscular development and nutrition. There was a swelling of the right knee and, on section into the joint, about an ounce of rusty brown fluid was removed. The bones were not fractured, though the joint tissues were swollen. The diagnosis was traumatic arthritis of the right knee.

The right femoral vein was filled with a thrombus and its wall was thickened, sclerotic and congested. Microscopic section showed the vein wall permeated by vasa vasorum which were surrounded by a perivascular infiltration of round cells and leucocytes. The left femoral vein was thin walled and normal. Figure 3 shows the contrast between the right and left femoral veins in this case.

The pulmonary artery was effectually blocked by a number of small thrombotic emboli which were the cause of death. Figure 4 discloses an embolus filling a large branch of the pulmonary artery. Figures 3 and 4 were photographed at the same magnification.

The uterus showed a papilloma about 1 cm. in diameter which projected into the cervical canal and evidently caused the uterine hemorrhage.

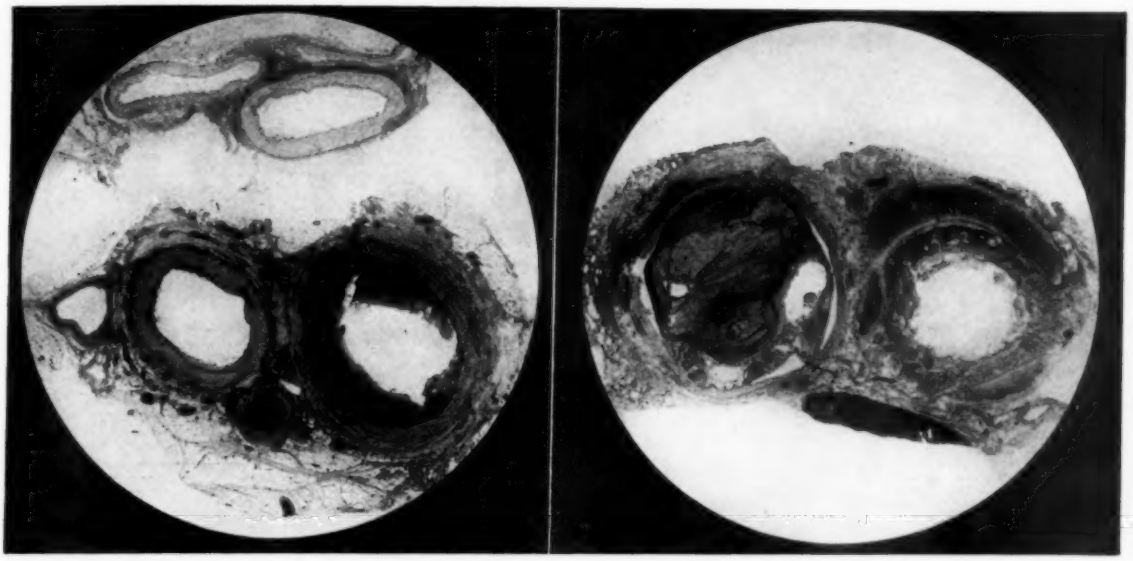

Fig. 3. Normal left femoral vein (upper left). Thrombosed thickened and inflamed right femoral vein (lower right).

Fig. 4. Embolus in pulmonary artery.

The other organs of the body were normal.

In this case the deceased in all probability had a mildly sclerotic femoral vein on the right side which antedated the accident. When her right knee was injured the trauma and the enforced inactivity caused an exacerbation of the phlebitis and the formation of a thrombus. The hemorrhage from the uterus because of the polyp doubtless altered the composition of the blood and encouraged the growth of the thrombus. Three weeks after the injury, parts of the thrombus detached themselves from the right femoral vein and produced a fatal pulmonary embolism.

The 30 cases of pulmonary embolism which were not associated with a definite traumatic lesion will not be discussed here in detail. It is sufficient to note that, while there was no trauma present, the pathologic lesions in the veins were not different essentially from those just described in the traumatic cases.

## SUMMARY

A summary of the important points brought out by the study of the cases associated with a definite traumatic lesion is as follows:

1. The sex incidence showed that 37 of the cases were women and 23 men.

2. The age incidence indicated that 50 per cent of the cases occurred between the ages of forty and sixty years.

3. The types of trauma which injured the patients were the ordinary casualties encountered in any large city. Of the injuries, 72 per cent were produced by automobile accidents. The lesions varied in severity from severe fractures of the pelvis or of the shaft of the femur to mere contusions and abrasions of the extremities.

4. The interval which elapsed between the onset of the trauma and the sudden death by pulmonary embolism varied between four days for the minimum and fifty-four days for the maximum. The average interval occurred between ten days and twenty-four days. This indicates that the influence of the trauma on the production of the thrombosis and the embolism was not similar in every case.

5. Where the trauma involved the bones and soft parts of one of the lower extremities, thrombosis was found in the veins of the corresponding lower extremity in 32 instances. In 14 examples of this type a thrombus could not be found. There were 14 cases of injury in which some other part of the body besides the lower extremities were involved; in 9 of these cases femoral vein thromboses were found. These figures suggest that a traumatic lesion near the femoral vein or its tributaries undoubtedly has an influence on the production of thrombosis in that vein. On the other hand, a similar thrombosis occurred in the femoral veins in injuries which did not affect the lower extremities and in such instances the influence of the trauma must have been more remote. In addition, femoral vein thromboses and fatal pulmonary embolism occurred in non-traumatic cases of all sorts, so that the basic etiology of this condition is some factor besides physical injury.

6. Microscopic investigation of the thrombosed veins of the lower extremity, both in the traumatic and the non-traumatic cases, disclosed that in almost all instances there was a phlebosclerosis, a subacute phlebitis or a periphlebitis of greater or less degree present. In many cases these lesions probably antedated the trauma. In others the effects of the trauma, whether general or local, probably precipitated the inflammation and produced a non-septic thrombosis which eventually resulted in a fatal pulmonary embolism. In our series it is impossible to interpret the course of events more exactly or to tell just how the three factors mentioned by McCartney operate in any one given case.

7. It is interesting to note that in 17 of the 21 cases which I examined personally, various acute and chronic disease conditions were present, which may have contributed to the formation of the thrombosis in the veins of the lower extremity.

## COMMENT

It is evident that thrombosis of the veins of the lower extremity followed by pulmonary embolism is a condition which must be considered by the surgeon as a possible complication of fractures and other traumatic lesions, especially in individuals who have reached middle age. The development of the thrombosis is insidious and the patient seems to be on the road to recovery; then without warning the pulmonary embolism develops and the patient dies suddenly in an attack of acute asphyxia.

Deaths by pulmonary embolism raise many interesting medicolegal tangles, especially in accident cases. There is always room for a difference of opinion concerning the part which the trauma has played in producing this complication. Obviously a bruise of the leg is not as serious an injury as a fracture of the lower third of the shaft of the femur, but both types may develop a pulmonary embolism. Perhaps the soundest view to take is to regard the trauma as the factor which precipitates the process. The basic causative factor is a vein of the lower extremity which is either diseased or a locus resistentis minorae and so is prone to develop a thrombophlebitis readily under slight provocation.

The author wishes to thank Dr. Charles Norris for the cases reported in this paper and Mr. W. J. O'Neill for the photographs.

# THE CLINICAL DIAGNOSIS OF FAT EMBOLISM

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FAT embolism can be defined as the introduction of liquid fat into the circulation and its transportation by the blood stream to occlude the finer blood vessels in various parts of the body. This condition is a frequent result of fractures of the long bones or any other trauma which is associated with damage to adipose tissue. In the majority of cases only an insignificant grade of fat embolism is produced by the types of injury mentioned. In a few instances, however, especially when the long bones of the lower extremity are fractured, a large quantity of fat may gain access to the blood vessels and so interfere with the blood supply of the vital organs that death results.

In most fractures of the long bones three factors are present which promote the development of fat embolism. They are: (1) The destruction of fat cells in the bone marrow and in the surrounding intermuscular tissues so that a large amount of liquid oil is produced. (2) The tearing of the venules in the Haversian canals of the bones which cannot collapse readily because of the rigid walls in which they are encased. (3) A mechanism such as muscular spasm or manipulation of the broken bones which increases internal pressure in the region of the fracture and so favors entrance of the fat into the veins.

In the majority of fractures of the lower extremities (80 per cent) the processes of repair are effective and prevent the entrance of any considerable quantity of fat into the blood stream. For this reason, serious cases of fat embolism are not common and when they occur it is probable that mechanical interference with the broken bones has interfered with the processes of repair.[1]

The way in which fat embolism develops in the human subject is not known in all its details. Many have the conception that the fat enters the blood as a coherent column of oil which blocks the pulmonary artery and causes immediate asphyxia. This eventuality may occur in rare instances, but in the vast majority of cases it is more probable that the fat is forced into the veins at intervals as insults of various sorts are offered to the injured extremity. Symptoms are produced when sufficient emboli accumulate in the lungs to impede the pulmonary circulation.

Clinically the serious and fatal cases of fat embolism are separated into two types:

1. *Pulmonary Fat Embolism.* In this type the fat is transported through the veins, the right heart, the pulmonary artery, and finally lodges in the arterioles and capillaries of the lungs. The flow of the pulmonary circulation is impeded and the patient shows signs of asphyxia.

2. *Systemic or Cerebral Fat Embolism.* In this type the fat emboli in the pulmonary capillaries are forced by pressure in the right side of the heart into the pulmonary veins, the left side of the heart, and the aorta, whence they are distributed all over the body. Most of these emboli reach the brain and cause definite damage of the brain substance. Because of this the patient develops a coma which becomes deeper and deeper until his death.

## CASE OF PULMONARY FAT EMBOLISM

CASE 1. The patient, a healthy adult white male, thirty-eight years of age, was struck by an automobile traveling at a high rate of speed. He was admitted to a hospital immediately

after the accident. A fracture of the right tibia and fibula was found which was compound in the mid-point of the shin region anteriorly by an opening ¾ inch in diameter. The patient was in deep coma with incessant tonic spasms of arms and legs.

Seven hours after admission blood began to ooze from the laceration of the skin at the site of the fracture. A pressure bandage was applied at this area to stop the bleeding.

One hour later the patient began to breathe rapidly at the rate of 44 to the minute, his pulse rate increased to 155 beats per minute, and his temperature rose to 107°F. Blood was present in the spinal fluid.

The next day the patient showed a respiratory rate of 52 per minute, an occasional cough, and distinct signs of pulmonary edema. The pulse rate had increased to 190 per minute. His coma had become much deeper. Death occurred twenty-nine hours after admission to the hospital.

The clinical diagnosis was: Fracture of the right tibia and fibula; possible fracture of the skull.

The necropsy was performed six hours postmortem. It disclosed a muscular adult white male, 5 feet 8 inches in height, weighing about 165 pounds. The compound fracture of the right tibia and fibula was found as described.

The scalp and skull were not injured. The brain showed patchy hemorrhages in the subarachnoid space over the outer surface of the cerebral cortex. Adjacent to these were numerous contusions in the gray matter of the cortex, 2 to 3 cm. in diameter, especially over the frontal lobes and the Sylvian fissure; there were some in the basal nuclei of the cerebrum and also in the corpus callosum. The contusions were not present in the rest of the white matter or in the cerebellum, pons or medulla. The brain showed only a slight degree of cerebral compression.

The lungs were free, congested and moderately cyanotic in appearance. A little atelectasis was present posteriorly. The right lung weighed 650 gm., the left weighed 600 gm.

The heart weighed 400 gm. and contained fluid blood. There was no foramen ovale.

The other organs were normal in appearance.

*Microscopic Examination:* Sections of the lungs, kidney and brain were stained for fat, using Sudan III and osmic acid.

Lungs: All parts of the lungs showed fat emboli plugging the arterioles and capillaries to such an extent that the alveolar walls were marked out by the globules of fat in the vessels. The lung was congested and edematous. Slight peribronchial bronchopneumonia was present. (Fig. 1.)

Kidneys: Numerous fat emboli were found in the glomeruli of the kidneys, but the kidney parenchyma was not damaged.

Brain: Fat emboli were found here and there in the cerebral cortex, but there were not any lesions present which were caused by them. The multiple contusions on the surface of the brain were not connected with the fat embolism.

*Comment:* In this case the deceased suffered from two distinct conditions, both of which resulted from the automobile accident.

First, there were the multiple contusions of the brain, caused by the severe impact of the body against the automobile. This violence did not injure the skin of the head or fracture the skull, but it did produce a vigorous oscillation and bruising of the brain inside the skull. The resulting contusions of the brain gave rise to the coma, the muscular spasm and a certain amount of cerebral compression.

Second, there was a pulmonary fat embolism which was the result of the compound fracture of the right tibia and fibula. The fat embolism was the immediate cause of death though the muscular spasms produced by the cerebral contusions enhanced the severity of the embolism. The contraction of the muscles around the site of the fracture raised the internal pressure in this region, caused the bleeding from the laceration over the fracture and forced fat emboli into the blood. The attempt to control the hemorrhage by a pressure bandage only succeeded in forcing more emboli into the blood. As a result the pulmonary capillaries were engorged with emboli, and the patient died of asphyxia about thirty hours after the trauma. The fat embolism in the brain and kidneys in this case did not have any appreciable effect clinically, but only

indicated the abnormal pressure in the pulmonary circulation which forced some of the fat from the lungs into the arterial system.

The clinical features of this case correspond with one of the varieties of pulmonary fat embolism described by Groendahl.[2] He recognizes two forms: (1) The type in which the patient dies suddenly a few hours after the injury in severe dyspnea, cyanosis and pulmonary edema. This is caused by the fact that a large amount of fat enters the circulation and causes a violent and sudden asphyxia.* This type is rare clinically. (2) The type in which the onset is more gradual and the symptoms come on many hours after the trauma. The fat emboli enter the circulation at intervals and accumulate in the lungs until the pulmonary circulation is impeded by their presence. The patient shows signs of increasing dyspnea and finally dies of asphyxia after an interval of a few hours. The case described here as well as Cases IV and V described in a previous paper[1] were of this type.

The importance of pulmonary fat embolism as a complication of fractures should not be overlooked inasmuch as the embarrassment of the pulmonary circulation may prove fatal to a patient who is suffering from a severe grade of surgical shock or who has a chronic cardiac, pulmonary or renal disease.

### SYSTEMIC OR CEREBRAL FAT EMBOLISM

In most of the cases of cerebral fat embolism the fat is forced from the lung capillaries into the left heart and the arterial system, whence it is transported to the brain, heart, kidneys and other organs. In some instances a patent foramen ovale may be present and it is conceivable that some of the fat may pass through this opening from the right heart into the left heart without entering the lungs. Most systemic emboli, however, pass through the pulmonary circulation and a severe systemic embolism may occur without a patent foramen ovale.

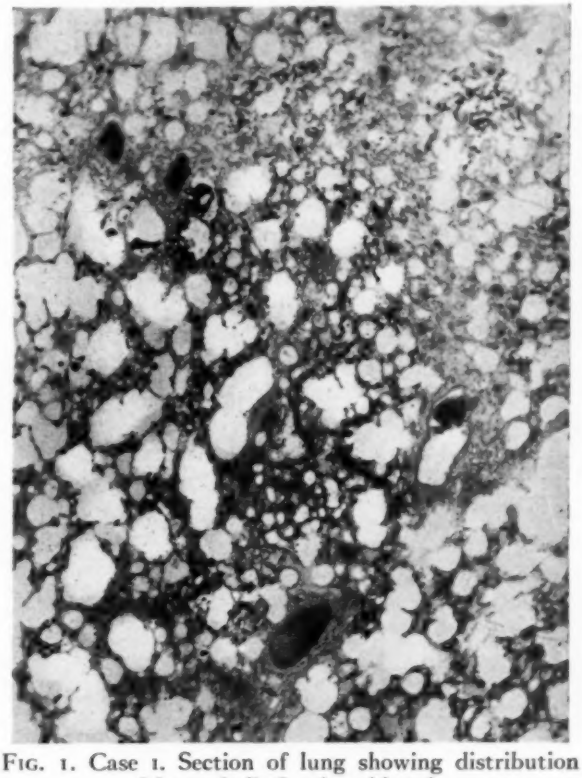

FIG. 1. Case 1. Section of lung showing distribution of fat emboli. Osmic acid stain.

The following cases illustrate the characteristic clinical picture of systemic or cerebral fat embolism. In the descriptions the clinical histories and macroscopic findings will be given for each individual case, but the microscopic reports will be given jointly to save needless repetition.

CASE II. A white man, fifty-seven years of age, medium height, obese, was admitted in cerebral concussion suffering from a fracture of the right femur, laceration of the left upper eyelid, and acute alcoholism. The history was that he had been struck by an automobile.

* Since this article was written one of my colleagues, Doctor Helpern, saw the following case which is a good example of rapidly fatal pulmonary fat embolism. A white man, fifty-four years of age, was struck by an automobile, sustaining a severe fracture of the left tibia and fibula and a slight fracture of the skull. About an hour and a half after the injury he appeared to be resting comfortably but suddenly he became cyanotic and died of acute asphyxia in a few minutes. The necropsy disclosed the injuries already mentioned and a marked emphysema, congestion and edema of the lungs. Microscopic examination of the lung showed that a severe capillary fat embolism was present, of sufficient grade to cause death by acute asphyxia. There was not any other organic condition or traumatic complication which could have caused death.

Eleven hours after admission a Thomas splint was applied to the right leg to immobilize the fracture.

The patient remained unconscious for two days, but on the second day the intensity of the coma lessened so that he was able to respond to his name. About that time his respirations became rapid and shallow. On the third day he lapsed back into coma again. The neurologic examination disclosed that he had a definite flaccid paralysis of the left arm and the left leg and a dilated pupil on the left side. The spinal fluid was bloody. A diagnosis of fracture of the skull and intracranial hemorrhage was made and a craniotomy was considered as a measure to relieve the cerebral symptoms. The poor physical condition of the patient, however, discouraged the execution of this idea. The patient sank deeper and deeper into coma and finally died five days after the injury.

At necropsy, a transverse fracture of the right femur at the junction of the middle and lower third was found with considerable injury of the surrounding parts.

There was a laceration of the left eyelid, a small fracture of the skull confined to the left orbit, and a small direct laceration of the brain adjacent to the fracture. These injuries were not particularly serious and were merely indications of where the force which produced the cerebral concussion had been applied to the head.

There was macroscopic evidence of fat embolism in the form of small petechial hemorrhages grouped in colonies of four to ten in the dermis of the upper anterior chest and shoulders. The white matter of the brain showed the same type of petechial hemorrhages grouped in colonies of four to ten. There were no hemorrhages in the gray matter. There were small streaky hemorrhages in the heart muscle. The heart did not have a foramen ovale. The lungs were large and voluminous, showing the usual patchy congestion and cyanosis seen in asphyxial conditions.

Histologic examination disclosed a widespread fat embolism in the heart, lungs, kidneys and brain. The brain tissue was definitely damaged by the embolic process, a point which will be discussed later in more detail.

*Comment:* The clinical history in this case illustrates many of the important features of cerebral fat embolism. The patient was admitted comatose from a head injury and in shock from a severe fracture of the right femur. His first twenty-four hours were spent in recovering from the cerebral concussion and, when he was just on the verge of regaining consciousness, he became dyspneic for a short period and lapsed back into coma. What probably happened at this point was that the fat emboli were blocking the pulmonary capillaries and then were forced into the arterial circulation by the right ventricle; they were carried to the brain, caused damage of the brain tissue, and produced the coma and paralyses already mentioned. These signs led to an erroneous diagnosis of intracranial hemorrhage following fracture of the skull, but fortunately an operation was not attempted.

The cerebral fat embolism in this case must be considered as the main cause of death.

Case III. A strongly built adult white male was injured in an auto collision, receiving a compound fracture of the left tibia and fibula, about 5 inches above the ankle. On admission, he was alcoholic, but conscious. A plaster cast was put around the fractured leg. Twenty hours later he lapsed into coma and finally died in coma forty-six hours after the injury.

The necropsy revealed that the fracture already mentioned was the only injury that he had received in the accident. There was obvious evidence of a generalized fat embolism, however, in the shape of innumerable petechial hemorrhages in the skin of the anterior portion of the neck, chest and shoulders, in the conjunctivae, in the white matter of the brain, in the lungs, bladder, heart muscle, larynx and large intestine. As in the previous case, the hemorrhages tended to arrange themselves in small groups of three to ten. There was no patent foramen ovale in the heart. Microscopic examination showed the presence of fat emboli in the various organs.

In this case the fat embolism was the sole cause of death.

Case IV. A white man, thirty-seven years of age, was injured in an automobile accident, dying three days later in coma. Further history could not be obtained.

The necropsy disclosed a muscular adult white male who had fractures of the left tibia

and fibula, the right tibia and fibula, and the left humerus. Section of the head showed a fracture of the left anterior and middle fossae and an acute suppurative leptomeningitis. There were numerous petechial hemorrhages of the type already described in Cases II and III, in the skin of the shoulders and neck, in the brain, heart muscle and conjunctivae. Microscopic examination disclosed the presence of a widespread fat embolism.

The fat embolism and the inflammation of the meninges probably contributed equally to cause the death of the patient.

Case V. A young white male, about nineteen years of age, was injured in a motor cycle collision, and sustained a fracture of the left femur. Sixteen hours after the injury he went into coma and developed paralyses and abnormal reflexes on both sides. He died in coma fifty-five hours after the injury. Fat embolism was suspected by the clinicians.

Necropsy disclosed that the death was caused by widespread fat embolism. The deceased showed the characteristic petechial hemorrhages over the skin of the shoulders and chest, in the conjunctivae, in the brain, bladder and heart muscle. A foramen ovale was present in the auricular septum. Microscopic examination of the various organs confirmed the diagnosis.

The *microscopic examination* of the organs was practically the same for all four cases.

The lungs disclosed a large number of emboli, but not as numerous as in the case of pulmonary fat embolism. The arterioles were plugged with fat globules and here and there some of the capillaries were involved. There was not any other finding of importance in the lung sections.

The kidneys invariably showed numerous coiled fat emboli in the glomeruli and sometimes in the afferent vessels, but the kidney parenchyma was not affected.

In the heart muscle the emboli appeared as ovoid globules in the smaller vessels between the muscle fibers. The adjacent muscle fibers showed fatty degeneration, each individual fiber being filled homogeneously with fine fat granules. In some instances red blood cells were infiltrated between the fibers, and the area of embolism was hemorrhagic. The lesions in the heart had all the characteristics of a microscopic infarct.

The brain showed numerous emboli in the vessels of the gray matter, but there was no change in the surrounding brain tissue. There were also numerous emboli in the white matter and these were accompanied by microscopic lesions in the brain substance. These lesions were of two varieties:

(1) The first was the ring hemorrhage which in effect was a microscopic hemorrhagic infarct. A cross section of the lesion showed in the center a small vessel plugged with a fat embolus and around the vessel was a zone of necrotic brain tissue. A zone of extravasated red blood cells surrounded the zone of necrotic brain tissue, forming a ring from which the name was derived. (Fig. 2.)

(2) The second was the area of rarefaction, a clear space in the brain substance caused by destruction and absorption of the medullary sheaths in the nerve tissue. These lesions showed numerous fat emboli in the vicinity which induced the necrosis. They are substantially microscopic anemic infarcts. (Fig. 3.)

The ring hemorrhages and the areas of rarefaction occurred in different proportions in the different cases, but their combined numbers were sufficient to account for the predominant cerebral symptoms. (Fig. 4.)

The petechial hemorrhages in the skin of the front of the shoulders, chest and neck were not examined microscopically in this series, but in a previous case the microscopic examination disclosed that they were due to a fat embolus in a small vessel of the skin which in some way caused an extravasation of blood into the loose tissues of the dermis. Just why they should be restricted to these particular areas is not clear, except that the blood supply is furnished by the subclavian arteries and these vessels receive large numbers of fat emboli. In addition, the dermis in this

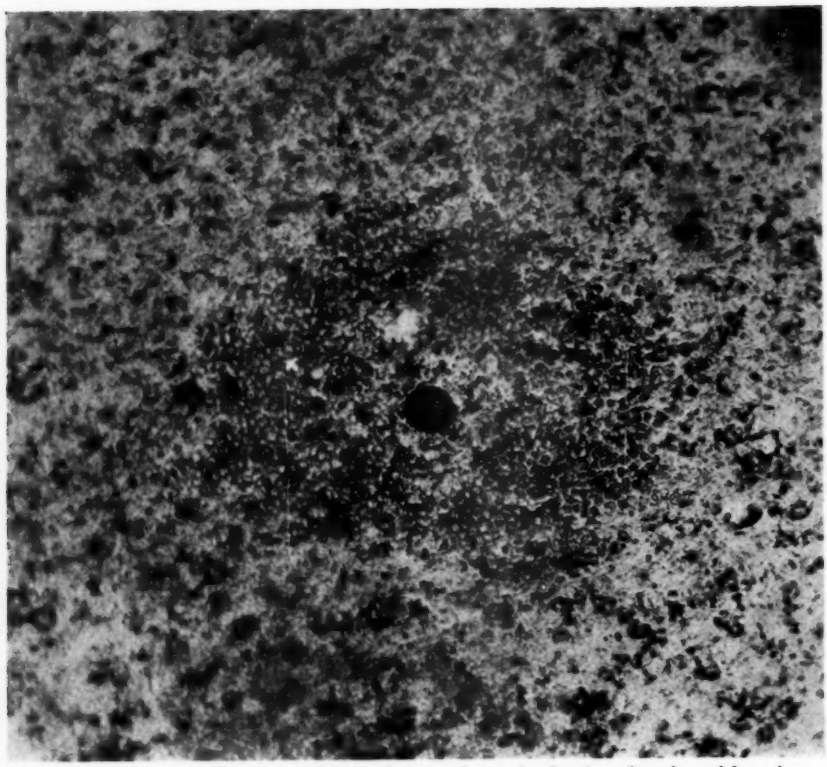

FIG. 2. Case II. Section of ring hemorrhage in brain. Osmic acid stain.

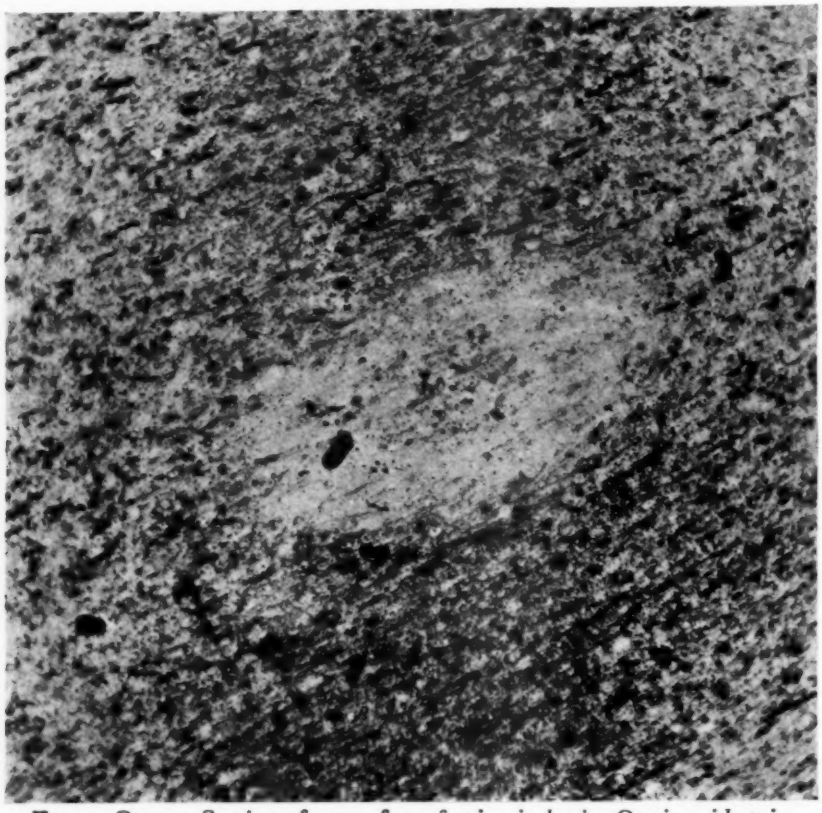

FIG. 3. Case II. Section of area of rarefaction in brain. Osmic acid stain.

area is not as fibrous as it is elsewhere and this may be a factor in the causation of the hemorrhages.

*Comment:* A review of Cases II, III, IV and V indicates that the clinical picture of cerebral fat embolism is rather characteristic, and in general corresponds to the description of Groendahl. There is at the start a period of quiescence which in our series varied from sixteen hours to two days. In several instances the symptoms of a concurrent head injury obscured this period. At the end of the interval the patient would show a little dyspnea and would lapse into coma. Vagrant paralyses and abnormal reflexes would appear, more or less unsystematized. When the coma started crops of petechial hemorrhages would appear on the chest, shoulders and neck, as described. All these signs were caused by the fat emboli invading the blood vessels of the brain and the skin and causing actual damage of these tissues. The heart, kidneys and other organs were also invaded, but it was not evident that their involvement produced any noteworthy symptoms. Death appeared to be due entirely to the lesions of the brain.

### DIFFERENTIAL DIAGNOSIS

The diagnosis of fat embolism is seldom made on the living patient because the condition is a comparatively rare one. Few clinicians encounter it in practice and a large number are prone to take the opinion that it is of slight importance. For this reason most of the diagnoses are made at the autopsy table.

Fat embolism should be considered as a possibility in every case of trauma associated with fracture of a long bone of the lower extremity. In the first two days following such an injury, any asphyxia or dyspnea should suggest pulmonary fat embolism as one of the likely diagnoses. Of course, a chronic cardiac or pulmonary disease would give practically the same symptoms and there is not any procedure except necropsy which could clear up this point.

The diagnosis of cerebral fat embolims can be made with much more certainty. The period of quiescence after the trauma

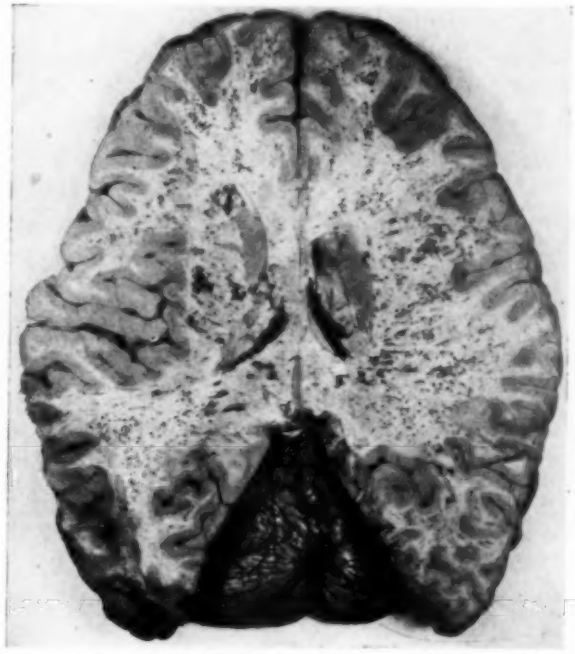

Fig. 4. Case V. Multiple petechial hemorrhages in white matter of brain.

and the lapse into coma coincident with the appearance of petechial hemorrhages over the front of the upper chest and neck, form a fairly characteristic syndrome. In some cases concurrent injuries of the head and extremities may obscure the clinical picture, especially if the petechial hemorrhages are absent, so that if coma occurs several hours after the trauma, it may be difficult to determine whether an intracranial injury or a cerebral fat embolism is present. In Case II this question actually did arise and only the poor physical condition of the patient prevented an exploratory craniotomy on a condition which could not have been relieved. Tobler[3] reported a case in which a craniotomy was performed on a case of cerebral fat embolism under the impression that the patient was suffering from an epidural hemorrhage consequent upon fracture of the skull. Possibly the chief distinctions between these conditions is that the gross traumatic intracranial hemorrhage is ac-

companied by a rise in intracranial pressure and the appearance of unilateral neurologic signs, while the cerebral fat embolism displays rather chaotic neurologic phenomena and not much increase in intracranial tension. The differential diagnosis, however, may be difficult unless the case is typical of one condition or the other.

Cases, some of which were reputed to be pulmonary fat embolism and some cerebral fat embolism, have been described in which the patient recovered. They are, however, of rare occurrence.[1]

There are many tests which are supposed to reveal the presence of fat emboli in the body, but they are not likely to afford the clinician much help. Large epithelioid cells containing fat granules in the spinal fluid and sputum were supposed to indicate fat emboli in the brain and lungs, respectively, but as the same cells are found in many inflammatory conditions not connected with fat embolism, their diagnostic value is nil. The presence of fat in the urine has been claimed by many to be a pathognomonic test of fat embolism, but it is not accepted by all observers. Fat emboli have been detected in the vessels of the retina by the use of the ophthalmoscope, and this test may be valuable in some cases in the hands of a competent observer (Oppenheimer,[4] Bernhardt[5]).

The treatment of fat embolism at the present time is to take all precautions to prevent its occurrence. As Case 1 indicates, the greatest factors in the production of this condition are disturbances at the site of the fracture in the early posttraumatic period. If these can be obviated, the embolism is less likely to develop.

The author wishes to thank Dr. Charles Norris for the cases here presented, Dr. Arthur M. Wright for two of the clinical histories, and Mr. W. J. O'Neill for the photographs.

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# OPERATION AND TRAUMA AS A CAUSE OF CORONARY AND CEREBRAL THROMBOSIS*

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IN recent years, a vast literature has accumulated relative to the occurrence of postoperative vascular thromboses. Particular attention has been devoted to changes in the blood-clotting constituents, in the hope of throwing light on the causation of this frequent postoperative tragedy. While a number of factors have been elucidated, the solution of the problems connected with postoperative thromboses is hardly nearer at hand than in Welch's[1] time, when he wrote his masterly monographs on thrombosis and embolism and explained the known factors in their causation.

It would seem, however, that insufficient attention has been paid to coronary and cerebral arterial thromboses as a postoperative complication. Indeed, so far as the former is concerned, the literature contains but a single case confirmed at autopsy, although Menard[2] has recorded 3 cases in which postoperative coronary occlusions were diagnosed clinically. Levine's[3] comprehensive monograph fails to mention the incidence of postoperative coronary occlusion at all from which it may be inferred that the condition either is rare or has been overlooked.

Four of the cases on which the present paper is based were obtained from the records of the Chief Medical Examiner's office and were autopsied by Dr. Benjamin Morgan Vance. It was Dr. Vance's feeling that these cases represented a definite group, in which trauma or operations acted as predisposing factors in precipitating arterial thromboses. The site of election of the thrombus was always a vessel previously damaged by disease, either arteriosclerotic or luetic in nature. Whether the trauma or operation acts upon the various clotting constituents in the blood to produce physical or chemical changes therein, or whether the primary cause is a degenerative change in the vessel wall to which the thrombosis is secondary, are questions which for the moment must remain unanswered. Bancroft and Stanley-Brown[4] emphasize the former viewpoint and in recent communications have pointed out changes in the blood clotting index which appeared to precede vascular thromboses. In the majority of their cases, however, the changes followed rather than preceded the thrombosis and it must be admitted that their work, however admirable, failed to point out any consistent general predisposing change in the clotting constituents in the blood which favored thrombosis.

Since, in every instance, the arterial occlusions occur in vessels that are already the site of previous damage and represent a site of lowered resistance, it seems more reasonable to suppose that the fundamental change is a degenerative one in a previously diseased vessel wall to which the occlusion is secondary.

## ACUTE POSTOPERATIVE CORONARY OCCLUSION FOLLOWING HERNIOTOMY

CASE 1. P. J. S., a fifty year old elevator operator, was admitted to the 4th Surgical Division of Bellevue Hospital, with a history of swelling of the left scrotum of six months' duration and a chronic cough with morning expectoration, for the past two years. He had had two previous operations for right inguinal hernia, the last one two years before at Presbyterian Hospital. He had received three months'

* From the Department of Pathology, Bellevue Hospital and the Office of the Chief Medical Examiner, New York City.

treatment for gonorrhea and chancre, fifteen years ago. About six months previous to admission, following a paroxysm of coughing, he had sudden pain in the region of the left scrotum, which thereafter enlarged. The patient's personal history was otherwise negative.

Physical examination showed a slightly cyanotic male, about forty-five years old, with a left oblique inguinal hernia and a right recurrent inguinal hernia. The chest was emphysematous in type. The heart was regular and there were no murmurs. Sounds were clear over the apex, but muffled over the base. In the lungs sibilant and sonorous rhonchi were heard anteriorly and posteriorly. The blood vessels were soft and compressible.

On admission, the blood pressure was 124/80; temperature was normal and the pulse rate was 76 per minute. Urinalysis and Wassermann test were negative. The clinical diagnoses were bilateral inguinal hernia, chronic bronchitis with emphysema.

The patient was operated on under spinal anesthesia (120 mg. of novocaine) at 11 A.M., for a right recurrent inguinal hernia and left scrotal hernia. Immediately after the operation, he complained of severe pain over the site of operation. At 6:30 P.M. (seven hours postoperative) his pulse became rapid and somewhat thready and he vomited a small amount of green fluid. He suddenly had a convulsion, accompanied by frothing at the mouth. Soon the patient became pulseless and cyanotic, breathing stertorously two or three times a minute. He received adrenalin intramuscularly and intracardially, followed by intramuscular caffeine and atropine. Respiration and pulse improved slightly. The patient had repeated convulsions at intervals of about fifteen minutes and died at 11 P.M. (twelve hours postoperative).

Necropsy was performed the next day, July 7, 1929, by Dr. Vance. The body was that of an adult white male, 5′ 5″ in length, weighing about 150 lbs. There were bilateral herniotomy incisions in the groins and an incision down the right thigh for removal of tensor fascia femoris.

On opening the abdomen, a laceration of the peritoneum was found in the left iliac fossa with a hematoma into the iliopsoas muscle. This was the source of a hemorrhage into the peritoneum, consisting of about 600 c.c. of current jelly coagulum. The source of the hemorrhage was a retroperitoneal vessel torn by a large Gallie suture.

The lungs were free, well aerated, with hypostasis posteriorly.

The heart weighed 310 gm. *The coronaries were moderately sclerosed. The anterior descending branch of the left coronary was blocked about 1 inch from the opening by a fresh thrombus, 1 cm. in length.* The valves and aorta were natural.

All the remaining organs were essentially natural.

The primary cause of death was coronary thrombosis, with abdominal hemorrhage, laceration of the peritoneum, resulting from a bilateral inguinal herniotomy.

*Comment.* A recent thrombosis of the left coronary artery appeared to have caused death in a fifty year old adult male, in apparent good health, operated upon for bilateral inguinal hernia. Among the possible predisposing factors are the spinal anesthesia, the intra-abdominal hemorrhage and the postoperative adrenalin.

## PAINLESS CORONARY OCCLUSION FOLLOWING CHOLECYSTOTOMY

Case II.* N. F., a fifty-three year old Italian taxi driver, was admitted to the 3rd Surgical Division with a complaint of pain in the right lower quadrant and vomiting of three days' duration. The case was diagnosed as acute cholecystitis and an exploratory laparotomy was decided upon.

Physical examination of the heart and lungs was negative. On admission the blood pressure was 127/72; pulse 80; respiration 20 and temperature 99–100°F. A red blood count was 6,000,000 with 100 per cent hemoglobin. White blood count was 14,000.

The patient was operated upon under spinal anesthesia, the day after admission. Operative diagnoses were: Biliary calculi and cholecystitis with empyema of the gall bladder. The operation performed was cholecystotomy with drainage. He was returned from the operating room after one hour and twenty-three minutes in fair condition with a blood pressure of 70/40 and a regular pulse.

Next day the patient perspired profusely and began to appear dyspneic and cyanotic.

*Case II is from the 3rd (New York University) Division, Surgical and Pathological Service, and is reported with permission of Dr. Arthur M. Wright.

The temperature rose sharply to 103°F., with a pulse of 128. No pain was present. There were palpable and audible rhonchi over both chests. On the second postoperative day, the patient's temperature rose to 105°F.; pulse was thready, rate 146, with no pulse deficit; respirations 40. He was moderately cyanotic, had a cough with tenacious sputum and resembled a patient with postoperative pneumonia. He then displayed signs of shock and pulmonary edema for which atropine was given. Later in the day, the patient became delirious. *X*-rays showed partial collapse of the lungs. The patient did not respond to energetic treatment and died forty hours after the operation.

Autopsy was limited to an abdominal incision. *Examination of the heart, which was moderately enlarged, showed an old well-organized thrombus in the left anterior descending branch of the coronary artery. The vessels were markedly sclerotic. Another smaller branch of the left descending coronary artery appeared to be completely occluded by an old process. Above the point of the old occlusion, these vessels were filled with soft, red, recent thrombi.* There was an old myocardial infarction with aneurysmal dilatation of the interventricular septum. The lungs showed some edema and were moderately collapsed. Chronic passive congestion was present in the liver. Kidneys showed a mild arteriolar nephrosclerosis. The ascending and descending aorta contained numerous calcified plaques and ulcerations.

Microscopic examination of the myocardium showed both old infarction, characterized by large areas of fibrous replacement and more recent areas of hemorrhagic softening. The appearance of the latter might well indicate an age of forty-eight hours.

*Comment.* A fifty-three year old male, operated upon under spinal anesthesia, died forty-eight hours after operation. At autopsy, both an old coronary occlusion, associated with infarction, and recent thrombi were found. The operation, of course, was in no way associated with the former, but the latter probably terminating event was undoubtedly a sequel thereto. The presence of a polycythemia and its association with arterial thrombosis are worthy of note as predisposing factors in the thrombosis.

### CORONARY OCCLUSION PROBABLY OCCURRING DURING COURSE OF OPERATION

Case III. A. S., a seventy year old houseworker, was admitted to the 1st Medical Division (P. & S.), with a complaint of fainting attacks for six years with moderate increase in dyspnea and orthopnea. Three days before admission the patient, for the first time, had noted substernal pain on exertion, relieved by complete rest. She had had high blood pressure for eight years, associated with moderate headaches, nocturia four to five times and attacks of palpitation of the heart.

Physical examination showed an obese, orthopneic, white female with face and forehead slightly flushed and cyanotic. Fundi were arteriosclerotic and eyes showed early cataracts. The chest was emphysematous in type, and the physical findings of emphysema were present in the lungs. On examination of the heart, an apex impulse was not felt. Borders were uncertain because of the emphysematous chest. Sounds were irregular with a blowing systolic murmur at the apex and base. There was an area of widened dullness over the manubrium. The aortic second sound was accentuated. Pulses were of high tension, the left slightly greater than the right. The abdomen showed no tenderness. Some slight pretibial pitting edema was present.

The patient, on admission, had a blood pressure of 175/120, which fell eight days later to 145/95. She had a low grade fever which could not be explained. Blood cultures, smears for malaria, Widal tests, melitenses, etc., were all negative. *X*-rays of the heart showed definite enlargement, transversely and longitudinally. Wassermann and urine examinations were negative. The pulse ranged from 80–100 and respirations averaged 20. On the twenty-first day, the patient was given 1 c.c. of salyrgan i.v. Fever was still of low grade and spiking. On the twenty-fourth day, she awakened with severe pain in the right leg, which was swollen, cyanotic and cold. The femoral pulse was scarcely perceptible. The impression was that of a thrombosis of the right iliac vein for which operation was decided upon.

At operation, under gas-oxygen-ether anesthesia, a large clot was found occluding the right iliac and femoral veins. The femoral artery was compressed and pulseless below the inguinal ligament, but following section

of the inguinal ligament, the arterial blood flow was restored. About this time, the patient ceased to breathe.

Necropsy was performed May 12, 1934, and showed the body to be that of an obese, elderly white female, approximately seventy years of age, 5′ 1½″ tall and weighing about 200 lbs. There was a recent freshly sutured right lower rectus incision, extending across Poupart's ligament. The right thigh was swollen and larger than the left thigh. On opening into the peritoneal cavity, about 50 c.c. of fresh blood were found in the pelvis. Intestines were in natural position.

On opening the pericardium, the pericardial sac contained about 60 c.c. of blood tinged fluid. Visceral and parietal pericardium were smooth and glistening. The heart was somewhat enlarged, weighing 500 gm. The right atrium was dilated about 1½ times the size of the left atrium. The ventricle was likewise dilated. The muscular wall of the left ventricle was hypertrophied and on section, showed scattered areas of fibrosis. The aortic valve showed atherosclerotic thickening of its leaflets and the coronary orifices were clear. The left coronary artery was patent throughout and showed fairly advanced coronary sclerosis. *The right coronary artery showed fairly advanced atherosclerotic changes. At a distance of about 5 cm. from the coronary orifice, there was a fresh thrombus, about 1 cm. in length, which completely occluded the lumen.* The heart muscles showed no gross or microscopic evidence of infarction. The aorta was inelastic and showed fairly advanced atherosclerosis with calcification.

The right iliac vein was occluded by a thrombus, which commenced about 4 inches from the entrance to the vena cava and extended about 1 inch below Poupart's ligament. The right femoral artery was almost occluded by a large atherosclerotic plaque, just at the level of Poupart's ligament. Both lungs were congested, as was the liver.

*Comment.* The author hesitates to assert that this coronary occlusion occurred during operation, since one cannot deny that the occlusion may have occurred unnoticed a day or two before death. No cardiac infarction was present, however, and there is no question that the thrombosis was of recent and probably operative occurrence.

### PAINLESS CORONARY OCCLUSION FOLLOWING INJURY TO THE FACE

Case IV. T. O'H., a thirty-eight year old male, had been struck in the mouth by an automobile door, about a week prior to death. The patient had gone home and later complained of severe headache and repeated vomiting. He was up and around for one week, but continued to have headaches, accompanied by nausea. The day before death he blew blood from his nose. Later, he became delirious and died suddenly in the presence of his family.

Autopsy on May 28, 1930, by Dr. Vance, showed an adult white male, 5′ 10½″ tall, weighing 168 lbs., and in a state of good nutrition.

On section, the abdominal cavity was natural. The lungs were heavy and edematous, but the bronchi and vessels were natural.

The heart weighed 440 gm. The left ventricle was distended with a large quantity of dark fluid blood. The right ventricle was partly contracted. The heart valves were natural. The foramen ovale was open as a slit, 1 cm. in diameter. *The left coronary was moderately sclerotic. The mouth and first centimeter of the right coronary were plugged by a shaggy, friable dumbbell-shaped thrombus. No cardiac infarction was present.*

*Comment.* Thrombosis of the right coronary artery occurred fatally, within one week, after a head trauma in a thirty-eight year old male, whose 440 gm. heart and moderately sclerotic coronary arteries suggested a pre-existing hypertension.

### POSTOPERATIVE AORTIC THROMBOSIS AND CEREBRAL EMBOLISM

Case V. I. R., a forty-three year old stone mason, was admitted to the 1st Surgical Division of Bellevue Hospital, with a chief complaint of a "broken right arm" for four months. The patient had fallen on a loose stone in the street, four months ago and had been unable to use the arm since. Seven months previously, he had been hit with a hammer over the upper third of the humerus, since which time the arm had always been painful. Six weeks before admission, he had noticed a lump over the right arm and had complained of pain in the left shoulder for twelve days. He had had slight

dyspnea on exertion, for the last four months. There was no weight loss. Personal history was negative and past health good, with the exception of bronchitis in the winters. Venereal disease was denied.

Physical examination showed a healthy, somewhat obese, middle-aged white male, with a barrel-shaped chest. The heart was enlarged slightly to the left of the mid-axillary line. Sounds were of good quality; rhythm was regular and there were no murmurs. The lungs were resonant and normal, except for some emphysematous breathing in the left lung, posteriorly. The remainder of the examination was negative.

On admission, the blood pressure was 155/80, temperature 98°F., pulse 80 and respirations 20. *X*-rays showed what appeared to be an osteogenic sarcoma of the right humerus. *X*-rays of the long bones and pelvis were negative.

He was operated upon under nitrous oxide and ether anesthesia. On incising the tumor a severe hemorrhage took place. The patient was in a state of postoperative shock, being pulseless, cold, clammy and unconscious. He did not respond to shock treatment of clyses of glucose, or to adrenalin series. A postoperative transfusion of 500 c.c. of whole blood was given. The following day, the patient went into a deep coma, breathing rapidly and irregularly. The pupils were contracted and equal. Heart tones were very rapid, pulses full and regular. All deep reflexes were absent. He was incontinent and perspiring freely. The temperature rose to 106°F. and the pulse rate was 160. On the second postoperative day, there was apparently no change in the patient's condition and he died at 9:45 A.M., forty-eight hours after operation, with a terminal rise in temperature to 107.6°F.

Urine was negative and Wassermann test of the blood was one plus. A postoperative red blood count was 1,760,000 with 60 per cent hemoglobin. A postoperative blood sugar showed 270 and non-protein nitrogen 45 mg. per 100 c.c.

Autopsy was on August 17, 1931, by Dr. Vance. The body was that of a well-developed adult white male, 5′ 5″, weighing about 140 lbs. Over the outer aspect of the right shoulder was a recent surgical incision, 4 inches long, involving the deltoid region and extending to the humerus. The latter had been hollowed out leaving a shell of bone.

On entering the abdominal cavity, the entire gastrointestinal tract, spleen, liver, pancreas, bladder and testes were natural, grossly. The lungs were well aerated anteriorly, but were markedly congested posteriorly.

The heart weighed 400 gm. The muscle was dark red-brown and flabby. Coronary arteries showed slight sclerosis. *The aorta showed a specific aortitis, extending from the aortic valves to the renal arteries, with a little calcification, plaque formation, thickened walls and longitudinal wrinkling. In the ascending aorta, near the valves, were small, fresh grayish-red thrombi attached to the intima, like sessile polyps. A few were present in the aortic arch.*

The right kidney was natural. The left kidney weighed 270 gm. At the lower pole there was an encapsulated hypernephroma, 1½ × 1 × 1 inches. It consisted of hemorrhagic, dark red, spongy tissue, mixed with a yellowish-gray, paler neoplastic growth.

On examining the head, the scalp showed a thick edema of the galea. The right cerebral hemisphere showed patches of red softening in the cortex and in the basal nuclei. A patch, 1 inch thick, was present in the left cortex involving the gray matter of the insula. The cerebellum and pons were natural and the vessels at the base showed no obvious thrombi.

*Comment.* A fresh thrombosis of the first part of the aorta, superimposed upon pre-existing luetic aortitis, resulted in fatal cerebral embolism in a forty-three year old man, operated upon for sarcoma of the humerus. The condition was in reality a metastasis from a hypernephroma. The thrombosis and embolism were of recent postoperative formation.

### POSTTRAUMATIC THROMBOSES OF RIGHT ANTERIOR AND MIDDLE CEREBRAL ARTERIES

Case VI. H. S., a forty-eight year old male, was admitted to an outside hospital, Feb. 8, 1928, complaining of pain in the head and abdomen, after having been struck by a taxicab three days previously. There were no apparent external injuries, but he died two hours after admission. Clinical impression was fracture of the skull.

Necropsy was performed February 8, 1928. It showed an adult white male, 5′ 3″, weighing

about 125 lbs., fairly well developed and nourished.

On section of the head, there was a slight contusion, the size of a silver dollar in the middle of the superficial layer of the left temporal muscle. On the right side of the brain there was a large area of softening, which involved the anterior convolutions from the vertex to just below the Sylvian fissure. The basal nuclei were patchily involved on the right. Vessels at the base were distended and showed only a few patches of sclerosis. In the right middle cerebral artery there was a thrombus, ¾ inch long, distending the vessel, just before and including the bifurcation. There was a corresponding area of softening in the right cerebral hemisphere. The right anterior cerebral artery was also involved in the thrombosis. The softened area was undoubtedly in its early stages, the convolutions appearing swollen and injected, contrasting with the paler gray convolutions on the side opposite and with the occipital convolutions on the same side. The lateral ventricles were slightly distended. There was no fracture of the skull, or injury to the brain.

The lungs were large and edematous, dark red from congestion and were both adherent to the chest wall posteriorly by fibrous adhesions. There were fractures of the seventh, eighth and ninth ribs, on the left.

The heart was slightly enlarged, weighing 400 gm. It showed flexible valves, natural musculature and coronaries with thickened walls and patchy sclerosis. The left ventricle was moderately distended.

*Comment.* A recent thrombosis of the right middle and anterior cerebral arteries causing fresh cerebral infarction, occurred in a forty-eight year old male, who had been struck with a taxicab three days earlier. The cerebral vessels showed a mild degree of sclerosis.

The foregoing group of cases, while not large, probably represents the largest single group of cases collected by a single observer of postoperative and post-traumatic arterial thrombosis, in which autopsy evidence is sufficiently complete to warrant the assumption that a definite causal relationship exists between the trauma of violence, or operation, and the occurrence of the occlusion. The coronary occlusions in Cases I–IV were all of recent appearance. All but one, Case II, were unassociated with cardiac infarction, which would corroborate the recentness of their formation.

Randall and Orr[5] recently reported 2 cases of postoperative coronary occlusion, one of which was confirmed at necropsy.

Case I was that of a fifty-eight year old man, operated upon under local beta-eucaine anesthetic for double inguinal hernia. The patient appeared to develop symptoms of beta-eucaine poisoning during the operation for which he was treated with adrenalin and intravenous infusion and died suddenly on the fourth postoperative day. At autopsy a white thrombus occluding the right coronary orifice was found. The description indicates infarctions of an area in the right ventricle.

Case II was a sixty year old man with a history of syphilis, who was operated upon for an epigastric hernia. Clinical evidence of acute coronary occlusion appeared on the eleventh postoperative day, followed by cerebral embolism on about the twelfth day. Unfortunately, electrocardiographic reading did not confirm the diagnosis and autopsy was not permitted. Hence, it is difficult to be assured of the time relations involved.

In their paper Randall and Orr quoted Wilson,[6] who mentioned one case of "coronary embolism" in a review of 47 cases of postoperative embolism following gastroenterostomies. Balfour[7] in a review of 57 deaths following gastric and duodenal operations listed one death as due to coronary thrombosis. Herrick[8] mentions a death from coronary thrombosis following cholecystectomy. Randall and Orr seem to have been the first to emphasize the probable causal relationship between operations and the fatal catastrophe.

Shortly thereafter, Menard and Hurxthal[2] reported the incidence of painless coronary thrombosis as a postoperative complication and reported 3 cases.

Case I was a fifty-two year old school teacher, operated upon for cholecystectomy, in whom coronary occlusion was diagnosed clinically

and corroborated with electrocardiographic findings and seemed to develop on the sixteenth postoperative day. Cerebral embolism developed. The patient made a partial recovery.

Case II was a thirty-two year old woman, operated upon for exophthalmic goiter. During operation circulatory collapse supervened and the patient was treated with adrenalin and oxygen. Electrocardiograph, shortly thereafter, showed inverted T waves characteristic of coronary thrombosis.

Case III was a fifty-six year old woman with enlarged heart and hypertension, who developed symptoms of acute heart failure on the fifth postoperative day. Electrocardiograph showed inverted T waves in leads I and II and a clinical diagnosis of coronary occlusion was made.

Although the clinical evidence was fairly complete in these cases, the absence of autopsy or clinical follow up records tends to render them somewhat less valuable as evidence.

There appears to be a prevalent clinical impression that various substances injected intravenously or intramuscularly are capable of precipitating coronary occlusions. Adrenalin and novocaine separately, or combined, are especially suspected in this regard. Weiss[9] has reported an example of fatal coronary occlusion following the use of novocaine and adrenalin for teeth extraction. The injection was attended by acute symptoms of precordial pain. Twenty days later, rupture of the heart occurred. Cardiac rupture usually occurs in the third week following occlusions.

Levine[3] mentions one case of coronary occlusion in which symptoms occurred during intravenous administration of iodine dye for cholecystography and states that he has seen three instances of this complication.

## SUMMARY AND CONCLUSION

Observations made in the postmortem room of Bellevue Hospital on material from the medical examiner's office and from clinical and autopsy material at Bellevue Hospital have convinced Dr. Vance and the author that there is a small though definite group of cases in which operations, or violent trauma, result in the occurrence of arterial occlusions in vessels that are the site of a chronic degenerative process.

Just what the mechanism of these thromboses is, the author is unable to state, but he wishes to point out that a definite clinical group exists in which fatal coronary or cerebral occlusions follow operations or violent trauma. Present surveys would indicate that the group is small, yet when without any particular effort, 4 cases of coronary occlusion are collected, 2 of which occurred in the past six months, it indicates that these cases are perhaps more frequent than is supposed and are often overlooked by the surgeon, who is not familiar with the medical diagnosis. Even the unsuspecting pathologist may overlook the diagnosis, because in all but one of the cases seen, cardiac infarction had not occurred and a small thrombus in a coronary vessel unassociated with infarction is readily overlooked.

Just what the precipitating factor or factors is, remains to be determined. Two of the operative cases were operated upon under spinal anesthesia. Cases I and IV received large dosages of adrenalin, postoperatively. The other cases received no adrenalin. In Case III the coronary occlusion appeared to have occurred during the operation.

None of the cases of coronary occlusion were attended by clinical symptoms of typical coronary pain and this perhaps explains why none of them was clinically suspected. Postoperative symptoms in Case I were probably referable to intra-abdominal hemorrhage and shock, the convulsions indicating cerebral anemia. Case II gave symptoms of acute left ventricular failure. Symptoms of coma and high fever in Case V suggest cerebral vascular accident as was actually found to be the case. Satisfactory history could not be obtained in Case VI.

The time interval between trauma, or operation and occlusion also appears to vary, but would appear to be rather short in all of our cases. In Case III, thrombosis appeared to occur during operation.

In Case I, thrombosis could not have been more than twelve hours old. The superimposed fresh thrombus in Case II was not more than forty-eight hours old. Thrombosis in Case IV was recent, undoubtedly less than a week old.

On the other hand, in Menard's[2] reported cases, thrombosis appeared to occur on the sixteenth day, during operation and on the fifth postoperative day respectively.

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# WOUNDS BY FIREARMS IN CIVIL LIFE*

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IN the investigation of projectile wounds of civil life, it is desirable and often necessary to correlate the observations of the primary investigation at the scene of a crime with those obtained at a subsequent necropsy. In this respect an inquiry into the circumstances, the character of the surroundings, and the preservation of various objects for future examination may prove useful.

Furthermore a subsequent examination of the deceased's clothing, the comparison of projectiles found at the scene with those recovered from the body, the investigation of associated weapons, cartridge cases, etc. may elicit data of indisputable value. And finally, a careful scrutiny of both the wounds and clothing preferably by a hand lens and perhaps a more detailed microscopic investigation of the surface lesions and tissues adjacent to the bullet track, may be desirable in occasional cases.

In general the examination of the clothing is properly the responsibility of the physician performing the necropsy, while the discharge effects of a suspected weapon in respect to the distance it was held from the clothing should be delegated to one thoroughly familiar with its limitations.

The examination of the projectile recovered from the body in relation to the rifle markings with a view to connecting it with a specific weapon, by comparison with a test bullet, may reveal important facts.[1] Likewise characteristic marks on cartridge cases, produced presumably by defects of the firing pin, ejector or breech block, and compared in a similar manner, may prove valuable.[2] Caution must be exercised in these interpretations however to avoid errors.

At times it may be necessary to determine the presence of blood, its type or grouping on the projectile, or the presence of other foreign substances such as pieces of clothing, hair, bone, etc., may deserve consideration.

Examination for marks and changes in form other than those produced by the rifling may reveal interesting if not valuable information. Among these may be mentioned those caused by defects in the barrel, reticulated patterns of the weave of clothing especially on the dome of soft lead bullets[3] (rarely observed on hard metal cased projectiles) various distortions and deformations, i.e. irregularities in shape, flattening, mushrooming, etc.

The splitting and breaking up of missiles due to contact with some hard substance (metal, bone, etc.) are relatively infrequent. When they do occur it is important to recover as many fragments as possible for the purpose of weighing them collectively in an effort to establish the calibre. Furthermore valuable facts may in rare instances be gleaned from careful scrutiny of each fragment.*

Where these distortions occur prior to entrance in the body, they may modify to a considerable extent the appearances

[1] BALTHAZAR, V. *Ann. de Méd. legale*, 3: 618, 1923.
GODDARD, C. H. *Army Ordnance*, p. 196 (Nov–Dec.) 1925. *J. Crim. Law & Criminol.*, p. 254 (Aug.) 1926.

[2] MAGE, J., and DE RECHTER, G. *Ann. de Méd. legale*, 3: 530, 1923. *Rev. de droit penal et de Criminol.*, 5: 126, 1925.
RAESTRUP, G. *Deutsche Ztschr. f. d. ges. gerichtl. Med.*, 7: 242, 1926.

[3] BALTHAZARD, V. Medicine legale. Paris, Bailliere, 1928, p. 274.

* A case in which some doubt arose whether we were dealing with a homocide or suicide, a hard metal cased bullet struck a lock manufacture by Sargeant of Indianapolis. Twenty one pieces were recovered and on two of them the letters S a r and i a n were noted.

* From the office of the Chief Medical Examiner of New York City

and other characters of the entrance wound (Fig. 1).

The examination of the surface lesions

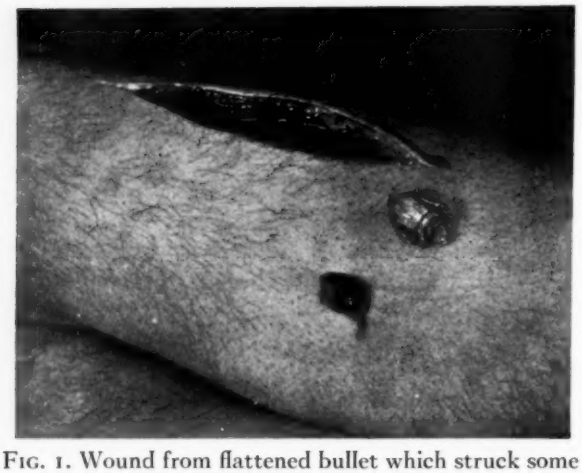

Fig. 1. Wound from flattened bullet which struck some object prior to entrance in leg.

and deeper track with a hand lens may elicit other important facts, such as the distinction between black and smokeless powders by the different color and form of any unburned grains which may be present,[4] and more rarely the detection of small spicules of lead torn from soft lead bullets by the rifling and found imbedded in the skin or clothing. Furthermore a chemical analysis of the powder residue if sufficient in quantity may reveal the difference between nitrocellulose and cordite powders.[5]

In this connection it appears important in the event the victim survives and is transported to a hospital, that the surgeon remove and preserve any powder residue, and incidentally bullets or fragments recovered at operation, in order to deliver them for proper examination.

The familiar leading and grease deposits on the clothing and about the edges of skin wounds may be mentioned to caution against confusing them with powder stains. They occur at all ranges and are of value in the differentiation between entrances and exits. Another source of error in this respect is the drying of the wound edges after several hours.

A further noteworthy observation is the microscopic detection of fragmentation or splitting of hair in the case of smokeless powder and the beaded appearance supposedly from black powder, thought to result from gasses formed in the medulla and cortex by heat.[6]

Finally besides the microscopic and chemical methods indicated above, spectroscopic and roentgenographic analyses have been recommended recently in the examination of entrances, tracks and exits for the detection of various metallic deposits.[7]

It is evident from these diversified methods of investigation that adequately equipped laboratories are indispensible and that close association of these with the department responsible for the investigations is most desirable.

The foregoing brief review refers in general to rifled arms, i.e., pistols, rifles and machine guns.

Shotguns deserve separate consideration.

## CHARACTER OF PROJECTILE WOUNDS

In order to fully appreciate the variety of surface and deep appearances of firearm wounds, a number of factors must be considered, namely, the character of the weapon (smooth bore or rifled), its calibre, type of powder, velocity of the projectiles, distance and angle of discharge, distortions of bullets before entering the body, influence of the clothing, etc. In the smooth bore in addition, the choke, number and size of shot and the wads deserve consideration.

Shotguns usually produce wound appearances greatly at variance with single shot weapons, while rifle and machine guns differ from pistol wounds by reason of their greater power and consequent increased destructive force.

[4] Smith, S., and Glaister, J. Recent Advances in Forensic Medicine. Phila., Blakiston, 1931, p. 60.

[5] *Idem*, p. 65.

[6] Hildebrand. In Lochte's Gerichtsärztliche und Polizeiärztliche Technik. Weisbaden, Bergman, 1914, p. 228.

[7] Gerlach, W. *Deutsche Ztschr. f. d. ges. gerichtl. Med.*, 22: 438 (Feb. 16) 1934.

In combination with the above desiderata, certain phenomena associated with the discharge of a gun are important, especially in respect to surface appearances; (1) the flame which produces the burn; (2) smoke (carbon) causing blackening or smudge, (3) unburned and slowly burning powder grains (tattooing), (4) the explosive force of suddenly released gasses at the muzzle, which cause the large gaping wounds in contact shots, and (5) the projectile.

Other less common effects are the previously mentioned spicules of lead blown from the muzzle and found imbedded in skin or clothing, and the somewhat more frequent metal fouling about the rim of the entrance wound in the skin or clothing.

The latter is often indistinguishable from grease and other deposits and may necessitate other methods of examination than direct inspection.[8]

All of these phenomena are based on the discharge mechanism which in modern arms may be summed up as follows: When the firing pin strikes the primer and the powder is ignited, a considerable amount of gas is formed causing increased breech pressure and forcing the bullet (which is slightly larger than the calibre of the weapon) through the barrel. A small proportion of the gasses escape about the bullet and emerge before it. The bulk of the gasses continue to exert force on the base of the projectile and emerge immediately after it leaves the muzzle. In its passage the bullet acquires a spin by virtue of the spirally arranged riflings. The purpose of this is to stabilize the missile in its flight and reduce air resistance, thereby increasing velocity, range and accuracy. This gyroscopic action causes the bullet to wobble for short distances in high powered weapons, such as the rifle, which accounts in some degree for the explosive effects of these weapons at comparatively short ranges (100 to 200 yards). Beyond these ranges the wounds differ little from pistol wounds except when soft nose, expansile or explosive bullets are used, and where a spent bullet gyrating irregularly strikes side-on, producing the familiar keyhole wound. The latter effect is rarely observed in small arm injuries except as a result of a ricochette or when the weapon or cartridge is defective.

In the further consideration of rifled projectile wounds, the angle of entrance may cause characteristic appearances. Ordinarily with the exception of contact wounds, a bullet striking at right angles to the skin surface produces a more or less round hole somewhat smaller than the calibre, the rim of which is surrounded by a collar-like zone of abrasion varying in thickness. This area results from the boring spin of the bullet and the abrasive action of its sides, as it enters stretching and indenting the skin. After the missile passes, the skin resumes its original position. This accounts for the discrepancy in size between the skin perforation and the calibre of the projectile, and the consequent impossibility of estimating the calibre from the dimensions of the skin wound.

When the wound site is unprotected by clothing, there may be in addition more or less metal fouling of the rim of the perforation, and of course powder effects in close shots.

At angles of 45 degrees, the missile may produce a beveled abrasion of the near side generally much greater in extent in comparison with a right angle wound, and as a rule the far edge is undermined. An entrance of this variety is often difficult to distinguish from an exit.

At greater angles the abrasive effect is more pronounced, until a slitlike wound or abraded area is the result. These are liable to be confused with a laceration or elongated abrasion (Fig. 2). When there is any doubt and the site is under clothing, an examination of the latter may assist by the presence of two or more holes, powder

[8] Eidlin, L. M. *Deutsche Ztschr. f. d. ges. gerichtl. Med.*, 22: 204 (Nov. 14) 1933.

residue and metal fouling about the entrance in the cloth.

Punctured wounds from round instruments such as a poker, ice pick, etc. may be difficult to distinguish clinically from a bullet entrance. There may be metallic staining of the edges similar to the metal fouling of projectiles.

Fig. 2. Abrasion of skin of thigh and entrance at an angle from a single shot.

### CONTACT WOUNDS

By contact wounds we refer to those produced by the muzzle held against the body.

Besides the large gaping irregular tears caused by the explosive effects of the suddenly released gasses at the muzzle, so noticeable when the injury is situated in a region with superficially placed bone covered by a comparatively thin layer of soft tissues, the presence of burned wound edges and interiors, smudge and undischarged powder grains in the depth of the wound serve to establish the diagnosis. These characteristics are quite common in head shots where the blackening may involve the inner and outer tables of the calvarium, the meninges and the brain substance.

Where this bone backing is lacking, as in the abdomen, some of the contact wounds may appear small and resemble more distant shots. The clothing may serve as a protection and the explosive force may diffuse in the weave of the cloth. Under these circumstances no difficulty need be experienced in view of the usual powder residue on the skin and in the depth of the wound, in association with like deposits on the clothing. Occasionally certain kinds of cloth (cotton, linen and silk) may become ignited and show large burned areas.

Contact wounds from rifles, machine guns and shotguns differ from pistol injuries in their greater destruction of soft parts and bone.

### WOUNDS FROM CLOSE SHOTS

A close shot may be arbitrarily defined as one in which the muzzle was held sufficiently close to cause discharge effects on the skin or clothing. They vary within wide limits depending upon the type of weapon, its calibre and the kind of powder. Individual weapons of the same calibre and make may differ in this respect; therefore no specific rule can apply. However, pistols using black powder may produce discharge effects up to and sometimes beyond a foot, rifles and machine guns 1 to 1½ yards and shot guns within 2 yards.

The large wounds so noticeable in contacts are ordinarily not observed in close shots. The latter differ little from more distant ones except in the varying degrees of surface burns, smudge and tattooing (Fig. 3).

It may be stated *a priori* that the greater the distance the less pronounced the discharge effects. At ½ inch the burn, smudge and a circumscribed zone of imbedded powder grains are practically limited to the edge of the wound and usually greater in extent above the injury provided the weapon was held in the upright position.

At greater distances these effects become less in intensity. The burn and smudge spread over a larger area and the powder grain wounds are more scattered.

At still greater ranges, depending on the kind of powder, the effects become less pronounced until the burn and smudge disappear and a widely scattered zone of powder grains remains the only sign of a

close shot. Beyond this the range cannot be estimated.

In respect to close shots, the range can only be approximated. The usual test of discharging a suspected weapon at blotting paper or similar substance may lead to erroneous conclusions. The texture, moisture and the circulation of the human skin is in no way comparable with this medium.

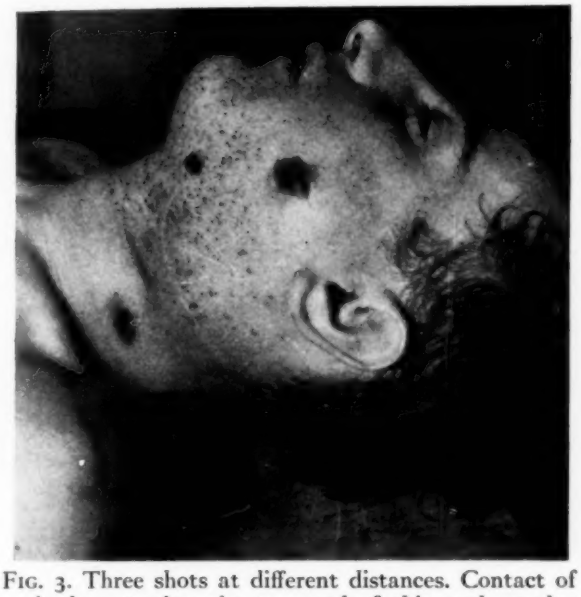

Fig. 3. Three shots at different distances. Contact of cheek, tattooing about wound of chin and smudge (blackening) above collar from close shot through it.

Similar experiments with clothing although more accurate can give only approximate results. In fact there are no methods known at present whereby one may accurately determine the range. All that can be testified to is whether we are dealing with a contact, close or distant shot and in close shots an approximation of the distance within reasonable limits.

A further difficulty is the distinct differences in the effects of black and smokeless powders. The latter may produce considerably less burn, smudge and tatooing by reason of more complete combustion. This is particularly noticeable on clothing (Fig. 4). Similar effects are observed in rifles and shotguns.

Guareschi[9] attempts to differentiate between the rifle and pistol by a combination of chemical and microscopic investigations of skin and clothing for the presence of mercury (from the fulminite of mercury in the primer) and endeavors to estimate the range. His observations indicate that a 7.65 mm. pistol gives positive results up to 5 cm. while a 9 mm. carbine gives results within 2.5 meters; considerably beyond the point where powder residue is demonstrable. He reasons from these experiments that in the absence of the customary powder stains on the clothing, the presence of mercury indicates the use of a rifle at a range between 1.5 to 2.5 meters. His experiments are open to the criticism that an insufficient variety of weapons were tested.

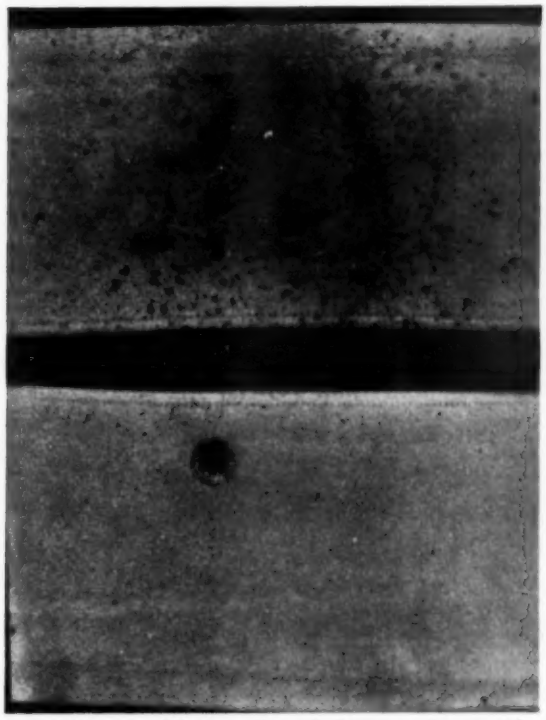

Fig. 4. Effects of black and smokeless powders at 6 inch range.

One effect not hitherto mentioned is the circumscribed burned areas, the result of slowly burning powder grains imbedded in the skin (Fig. 5).

## EXIT WOUNDS

Exit wounds of rifled arms vary greatly in appearance but have certain character-

[9] Guareschi, G. *Deutsche Ztschr. f. d. ges. gerichtl. Med.*, 23: 89 (Apr. 20) 1934.

istics which enable one to differentiate them from entrances in most instances.

They vary in size from a small slit, often

FIG. 5. Lesions caused by slowly burning powder grains. Note larger lesions above entrance and compare with fine stippling from unburned grains.

smaller than the entrance, to gaping everted irregular tears assuming various forms; sometimes round or oval, stellate, etc. The size of the wound is often influenced by distortions of the projectile, and from foreign substances carried along with the bullet, such as pieces of cloth, bone, etc.

Sometimes when the entrance and exit wounds are close together and the missile entered and emerged at an angle, it may be difficult to determine the entrance from the exit.

Occasionally the bullet is split on striking bone and may cause two or more exits. On other occasions part of the split bullet remains in the body while the other portion emerges. This possibility must be kept in mind in the clinical examination of these wounds. The use of the roentgen ray is indicated if doubt exists.

In head shots it is often possible, provided the cranium is not shattered, to distinguish an entrance from an exit by the sharp edged hole in the outer table and the beveling of the inner one. In an exit these characters are reversed.

### INJURIES FROM SMOOTH BORE GUNS

There should be no difficulty in the distinction of these from single shot injuries. By virtue of the number of shot in shotgun shells, which vary from several hundred in the smaller sizes to nine slugs in the ooo buck shot, the wads and the larger charge of powder, wounds from these weapons differ considerably from single shot injuries. Contact shots cause large irregular wounds with marked destruction of both soft parts and bone. The latter is usually extensively shattered. In head shots the cranial vault may be completely destroyed. Shots in cavities, as the mouth, may show extensive lacerations of the cheeks, shattered base of the skull and an irregular exit with marked destruction of brain tissue.

At ranges up to 3 feet with a choke bore weapon, a single large round or oval surface wound is the usual result, surrounded by a number of imbedded powder grains and more or less burn and smudge.

The intensity of these effects may vary depending upon the type of powder. With smokeless powders the smudge and tattooing may be negligible due to complete combustion in a long barrel weapon.

At greater ranges, 2 to 2½ yards, the central hole caused by the larger mass of shot is surrounded by a few perforations. There may be moderate powder stains or only a few unburned grains. In rare instances a sieve-like wound is observed, with some residue effects.

At still greater ranges the shot scatter and discrete wounds are the result. They may resemble multiple wounds from single shot weapons, especially if the larger size shot were used.

Although black powders produce more extensive effects, there seems to be no appreciable difference between bulk and dense smokeless powders.

In the modified choke and cylinder bores the shot scatter more freely and the wounds are consequently more widely

distributed at the same ranges in comparison with the choke bore.

The penetrating power of the shotgun varies according to the size of shot, powder charge and range. Small size shot at 40 yards may produce only superficial injuries while buck shot are considerably more destructive and penetrate more deeply. The danger of the superficial wounds lies in infection.

In close shots the wads may be found in the wound and besides their diagnostic value may disclose important information relative to the calibre, size of shot, type of powder and sometimes the manufacture of the ammunition. Wads are a potential source of infection if not discovered and removed.

The same may be said of the wads of blank cartridges where the danger of tetanus and other infections is fully appreciated.

The opinion that firearm missiles are sterilized by the heat of the discharge has not been proved and is of little importance. Foreign particles and bacteria may be carried in by the projectile and cause infection; which is not an infrequent cause of death in patients who survive the initial hemorrhage.

### RIFLE AND MACHINE GUN WOUNDS

Wounds from rifles and machine guns are more difficult to distinguish from pistol wounds. In contact and close shots the greater destruction, the larger area of burn, smudge and tattooing may help. With smokeless powder the same observations hold true with these weapons as shotguns in respect to the lesser degree of residue effects. When bullets are recovered there should be no difficulty in the diagnosis. Usually, at average ranges these missiles pass through both body and clothing and, when they do not strike bone, may produce wounds closely resembling pistol wounds. This holds true except at comparatively short distances when the previously mentioned explosive effects may be prominent.

The various methods of investigation noted above apply equally to wounds from these weapons.

### INTERNAL EFFECTS

When death ensues immediately or shortly after the receipt of a gunshot injury, in the majority of cases the cause is hemorrhage from injury to some part of the circulatory system.

Shots through the heart and the large blood vessels, i.e. aorta, pulmonary artery, vena cava, etc., are usually rapidly fatal. There are instances however when these patients may survive for a considerable time. Vance[10] reported a case of injury to the heart and aorta in which the patient survived eight days and died suddenly of secondary hemorrhage. Several cases have been observed of patients similarly shot by various calibre pistols, surviving for several minutes after receipt of the injury.

In general, wounds of the auricles are more rapidly fatal than those of the ventricles. Despite this, one patient lived for two days before secondary hemorrhage occurred.

In heart wounds, hemorrhage into the pericardium occasionally causes fatal tamponade of the heart. More frequently the blood leaks through a perforation in the pericardium into one or the other pleural cavity with consequent collapse of the lung.

Injuries of the coronaries are not especially frequent.

Wounds of the aorta causing slight tears may result in a dissecting aneurysm. The same applies to other vessels, and the fatal issue may be delayed for some time. This type of injury is rare and is comparable in this respect to the development of arteriovenous aneurysms from wounds of adjacent artery and vein.

Injuries to the innominate, subclavian, iliac, carotid and femoral arteries are usually rapidly fatal. Occasionally death is deferred long enough to permit operative procedures.

[10] Vance, B. M. *Am. J. M. Sc.*, 169:872 (June) 1925.

Shots through the lungs are generally followed by only moderate hemorrhage unless a large pulmonary vessel or the intercostals are involved. An added danger is the development of pneumothorax.

Injury in this situation may be followed by subcutaneous emphysema. Similarly a wound at the hilus may result in a mediastinal emphysema, subsequently spreading into the fascial planes of the neck and thence becoming generalized.

If, in the absence of these complications, infection does not supervene, recovery is not uncommon.

The dangers of chest wounds involving the heart and lungs when the patient survives the initial hemorrhage, may be summed up in secondary hemorrhage and infections of various types, i.e. general sepsis, pericarditis, septic pleuritis and empyema, pneumonias, lung abscess and gangrene and thrombotic processes.

In abdominal wounds, two types of organs must be considered: the solid and the hollow viscera.

When large blood vessels are injured within or without the organs, fatal intra-abdominal hemorrhage may arise. A similar result occurs when a number of small vessels are involved.

The surgical treatment of wounds of the liver is in general unsatisfactory largely from the difficulty of stopping the hemorrhage. Furthermore when the hemorrhage is negligible, injuries to the common bile duct and its branches in the liver and also the gall bladder, with consequent leakage of bile, may prove fatal from peritonitis or cholemia.

Another frequent result of liver wounds is the development of infarction along the track of the bullet manifested by light yellowish areas sharply demarkated from the normal liver tissue. In rare instances following infection an abscess may supervene.

Wounds of the spleen are fairly common and present no special points of interest. If other important organs have escaped injury, splenectomy may prove successful.

Wounds of the pancreas are peculiarly fatal by virtue of the escape of its ferments about the organ or in the peritoneal cavity and the production of a widespread fat necrosis.

Although situated extraperitoneally, the kidneys may be considered here. Wounds of these organs, if not quickly fatal, are prone to infection consequent on urinary extravasation. Bullet injuries here are somewhat more dangerous in comparison with injuries from blunt force by reason of the increased liability to infection.

Wounds of the hollow viscera are almost invariably fatal when neglected and often after operative procedures.

Occasionally injuries of the duodenum and upper intestinal tract with the escape of their contents (bile and pancreatic secretion) present a similar picture to wounds of the bile passages and pancreas, namely bile staining and fat necrosis, added to inflammatory changes of the peritoneum.

Injuries to the urinary bladder are not particularly infrequent and deserve the same consideration as other hollow viscera. The danger aside from hemorrhage is extravasation and infection.

Bullet wounds of the pelvic region for some unknown reason are peculiarly fatal and are usually attended with considerable shock.

Although in this brief review, projectile wounds of the various organs were discussed separately for convenience, injuries to a combination of viscera both solid and hollow are usually observed. This complicates the situation considerably and fine judgment must be exercised in relation to surgical procedures most suitable for the occasion. The question to wait or operate immediately is often difficult to decide.

Bullet wounds of the cervical region when the carotid vessels or the jugular veins are involved usually result in rapidly fatal hemorrhage. When, however, internal hemorrhage occurs through a wound in the larynx or trachea, death may be caused by

asphyxiation from aspiration of blood. Extensive bleeding may also occur in the subcutaneous tissues and fascial planes of the neck causing asphyxia by pressure on the pharynx, larynx or trachea.

In the event that the primary hemorrhage is negligible, subsequent infection, phlegmonous inflammation and glottic edema may cause death in a similar manner. When the larynx or trachea is injured, aspiration pneumonia, abscess and gangrene may develop.

Wounds of the pharynx and esophagus may prove fatal by excessive hemorrhage with swallowed blood in the stomach and aspirated blood in the lungs. Furthermore infection similar in character to the types just mentioned in laryngeal and tracheal wounds is not infrequent.

A fairly reliable sign of injury to the respiratory system in chest and neck wounds is the presence about the mouth and nose of blood-stained froth. It may also be noticeable in wounds of the pharynx and esophagus in rare instances.

Uncommonly, slight injuries to the carotid arteries or the jugular veins, insufficient to cause appreciable hemorrhage, may favor thrombotic processes and result respectively in the development of cerebral and pulmonary embolism. At other times injuries to the jugular, subclavian, innominate and other veins may allow the entrance of air and a fatal air embolism may result.

Head injuries which involve the cranial cavity and its contents in the majority of cases are rapidly fatal from excessive hemorrhage and brain laceration. Those patients who survive almost invariably become immediately unconscious on receipt of the injury and remain so until death. Sometimes, in the interim, a variety of symptoms and signs indicative of focal or general brain injury or compression or both present themselves. Uncommonly the patient may remain conscious for a time and subsequently develop symptoms of intracranial pressure from hemorrhage or edema. Infectious processes may supervene, i.e. leptomeningitis and pachymeningitis and rarely abscess. Recovery may occur in isolated instances usually with residual symptoms referable to the special senses, the cerebrospinal and uncommonly the autonomic nervous systems. Mental disturbances and epilepsy may develop or, in rare instances, recovery may be complete.

A not uncommon finding at necropsy in patients who have remained unconscious for several days is an aspiration bronchopneumonia, and when the bone sinuses (sphenoid, ethmoid, maxillary and frontal) are injured, aspirated blood in the lungs.

Wounds of the spinal column and cord are rarely immediately fatal with the exception of injury to the medulla and occasionally the upper cervical cord. The muscular and sensory paralysis below the level of the lesion may result from actual laceration, hemorrhage in the substance of the cord or into the membranes with consequent compression, and pressure from the missile itself.

The paralyses are likely to be complete when the cord is severed, likewise in medullary hemorrhage. Occasionally the fatal issue is ushered in by the development of pneumonia before the urinary tract infection and trophic disturbances occur.

At times although the projectile passes through the canal, it may miss the cord and cause little disturbance. The outlook in these cases may be favorable unless secondary hemorrhage or infection supervenes. When the missile remains in the canal it may produce subsequent cord changes. The presence of the bullet in the canal, although causing no symptoms, is always a potential source of danger.

Wounds of the extremities are usually of little moment unless a large vessel is injured or the bones or joints are shattered. The same danger in respect to infection applies here as elsewhere.

Concerning ricochettes: after the bullet enters the body it usually pursues a direct course. Occasionally when the projectile

strikes bone at a tangent, ricochettes may be noted. They are in general more frequently observed in the cranial cavity.

These phenomena require, in the conduct of the necropsy, that the bullet track be carefully followed, otherwise confusion may arise and an erroneous interpretation of the original direction of the shot may occur.

At times, antemortem or postmortem displacement of the bullet may resemble a ricochette. Failure to demonstrate a track from the original point of ricochette will assist in the differentiation.

A case presenting these features was that of an individual who was shot in the back at the level of the seventh dorsal segment with a 25 calibre automatic. He developed the usual paralysis and trophic disturbances and died one month after receipt of the injury. At the necropsy only a faint scar remained to indicate the wound of entrance. The cord was a soft pultaceous mass at the level of the injury. After some difficulty the bullet was discovered in the spinal canal at the level of the third lumbar vertebra, lying between the dura spinalis and the arachnoid. No track was demonstrable from the original injury to the situation of the missile.

Not infrequently a bullet will enter a blood vessel and be carried along in the blood stream. This occurred in one case, in which the missile entered the thoracic aorta and was found in the left iliac artery. A projectile may enter the pharynx and be discovered in the stomach; or it may enter some part of the intestinal tract and be evacuated in the feces.

All of these possibilities must be kept in mind. They increase the difficulties in the search for bullets, and often a roentgenographic examination is necessary.

In conclusion, it may be stated without fear of contradiction, that in addition to the necessary procedures already mentioned, the various immediate effects and subsequent results of projectile injuries must be appreciated in order to avoid misinterpretations and errors in antemortem and postmortem diagnosis. From a forensic viewpoint, in the conduct of the necropsy every effort should be made to determine the direction of the bullet tracks, to distinguish between entrance and exit wounds, to estimate approximately, if possible, the distance from which the weapon was discharged and the recovery of every bullet remaining in the body. For not infrequently guns of different calibres may be used.

Much may depend on these observations in establishing the innocence or guilt of a suspected individual.

# UNUSUAL FATAL STAB WOUNDS OF HEAD & NECK

## WITH EXAMPLES OF UNRECOGNIZED ICE PICK WOUNDS OF THE BRAIN*

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NUMEROUS and varied examples of fatal stab wounds are observed among the routine necropsies performed by the Office of the Chief Medical Examiner in New York City. In most of these cases, the wounds are of such obvious character, that no difficulty in their detection is encountered either clinically or at postmortem examination.

In this paper, attention is called to an unusual group of fatal penetrating stab wounds, especially of the head and brain, produced by long slender pointed weapons. In three of the cases to be described, the lesions were probably inflicted with an ice pick, a type of instrument which because of its availability and low cost, has recently gained popularity as a means of assault.

The small perforation on the surface of the body leading into the long slender tract of a stab wound produced by a weapon of this type may bleed very slightly or not at all, and when situated on the scalp and concealed by hair, may easily escape detection, or when located on the face, may be mistaken for a small superficial laceration. Should the skull and brain be penetrated, the resulting stupor or coma may make it impossible to elicit a history from the patient and if information from other sources is misleading or not obtainable, the clinical signs may readily be ascribed to other causes. When a person is found dead, after having been stabbed through the skull and brain with an ice pick or thin knife blade, the same difficulty in the detection of the wound may be encountered. In the absence of any suspicious surrounding circumstances, and without a complete and careful autopsy, the true cause of death may readily be overlooked. The circumstances in a case of this kind are easily altered and confused if the deceased, while still alive, is carried away or staggers away from the scene of the crime.

In addition to the three examples of ice pick wounds, a clinically undetected penetrating knife wound of the head and brain will be described, and also two cases of stab wounds of the neck and carotid arteries with mural thrombosis and cerebral embolism. A very unusual stab wound of the neck and superior mediastinum penetrating the left innominate vein and aorta, with the formation of an arteriovenous aneurysm, will also be described.

### CASE REPORTS

CASE 1. Penetrating stab wound of right side of head and brain. Homicidal assault with an ice pick. Duration of illness five days.

*Clinical Record.* E. G., a twenty-three year old negress, was brought to The Harlem Hospital July 19, 1933, several hours after an altercation, during which she was said to have been stabbed in the right temple with an ice pick. It was stated that there had been immediate loss of consciousness and considerable external hemorrhage from the wound.

On admission to the hospital, the patient was semi-comatose and acutely alcoholic. The head was sharply turned to the right and both eyeballs deviated to the right with a lateral nystagmus in that direction. The deep reflexes were hyperactive and no abnormal ones were observed. A small puncture wound which was not bleeding was noted in the right temporal region midway between the outer corner of the eye and the top of the ear. A blood-tinged spinal fluid under the increased pressure of 270 mm. was obtained by lumbar puncture. On examination seventeen hours later, the patient had

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regained consciousness, was able to mumble incoherently and attempted to cooperate in carrying out simple commands. A quadriplegia

The patient's condition remained unchanged for about twenty-four hours and following a short period of slight improvement, she be-

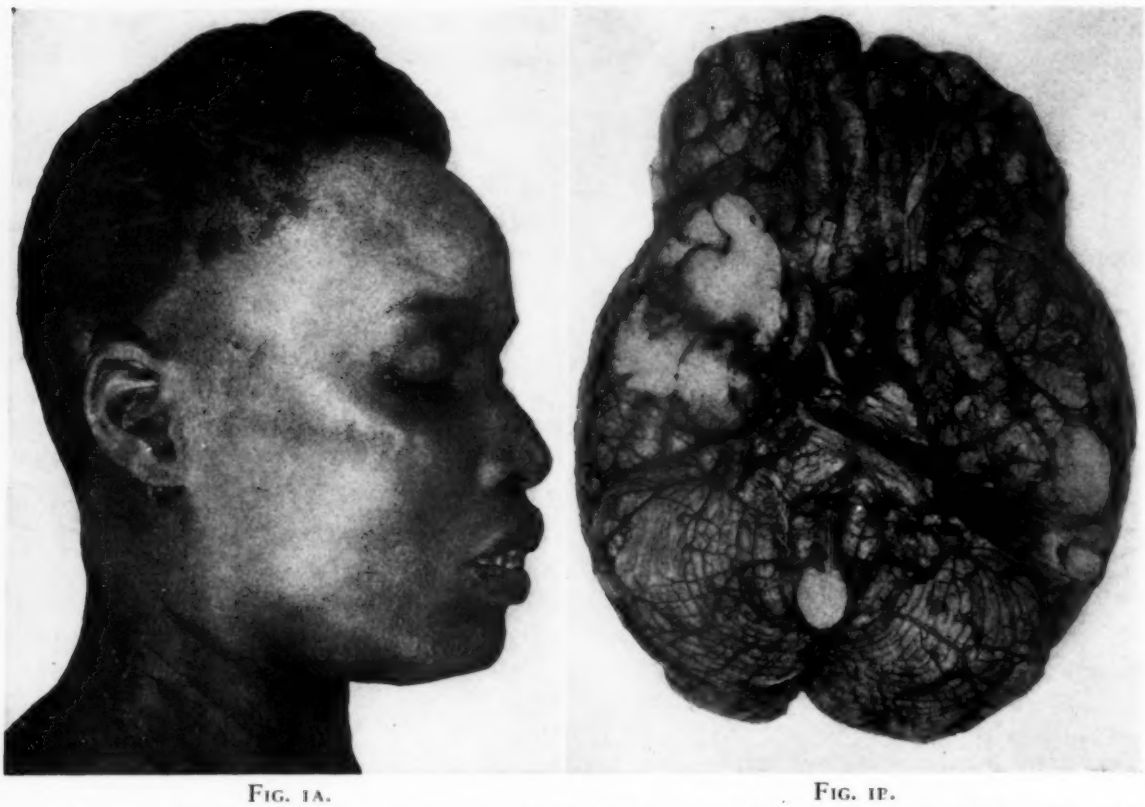

FIG. 1A. FIG. 1B.

FIG. 1. Case 1. A. Small surface lesion of a penetrating ice pick wound through right side of head. Temple has been shaved. B. Long slender tract of wound through brain. Tissue beneath tract has been cut away to show its extent.

with incomplete paralysis of the extremities on the right and complete paralysis of the extremities on the left was noted. The pupils were equal, 3 mm. in diameter and reacted to light. The fundi were normal. The eyeballs maintained the right lateral gaze. Deep reflexes were more active on the left than on the right. The plantar reflexes were normal and the ankle jerks were not elicited. There was a left lower facial weakness and the left side of the body was hyperalgesic. The blood pressure was 124 mm. Hg systolic, 76 mm. diastolic.

An *x*-ray picture of the skull revealed a puncture wound in the squamous portion of the right temporal bone. The blood sugar was 148 mg. per cent. The temperature fluctuated between 100° and 104°F., and the pulse rate was in proportion. Blood count: 9200 leucocytes, 68 per cent polymorphonuclears; 4,310,000 erythrocytes, 75 per cent Hb.

came worse and died five days after admission.

The clinical diagnosis was a puncture wound of the skull and lacerated wound of brain extending from the right side into the left cerebral hemisphere. Operation was considered inadvisable.

*Necropsy*. Anatomical Diagnosis: Penetrating stab wound of right temporal portion of scalp, right temporal muscle, squamous portion of right temporal bone, right temporosphenoidal lobe, right tentorium, pons, left cerebellar hemisphere, left tentorium, left occipital lobe, slight hemorrhage along tract, slight localized subdural hemorrhage, cerebral edema and compression.

A single small penetrating stab wound 1.5 mm. by 3 mm. with slightly abraded edges and covered with a soft brown scab, was found on the right temporal region of the scalp, 3 cm. posterior to the outer corner of the eyebrow,

and just above the level of the external auditory meatus (Fig. 1 A). The direction of the tract was from right to left backwards and slightly upwards. It passed through the temporal muscle 1.5 cm. above the zygoma and perforated the squamous portion of the temporal bone making a circular hole 3 mm. in diameter punched out on the outer table and beveled on the inner table. In this region the bone was 3 mm. thick. The dura was tense and the cerebral convolutions flattened. The pial blood vessels were congested. A small subarachnoid hemorrhage was distributed over the base of the brain. The tract of the stab wound across the brain was readily traced. Having pierced the dura, it traversed the right temporosphenoidal lobe entering on its lateral surface. It continued through the right tentorium and obliquely through the substance of the pons, then through the left cerebellar hemisphere, the left tentorium and the left occipital lobe which was penetrated through its inferior surface, ending 11.5 cm. from its entrance on the right side of the brain and 12.5 cm. from the wound in the scalp. Small localized subdural collections of blood were located around the holes in the tentoria and dura. The entrance and exit holes in the brain substance were small, measuring 3 to 5 mm. in diameter. After hardening the brain in formalin the tract was opened and only a small amount of hemorrhage was found along its contused slender course (Fig. 1 B).

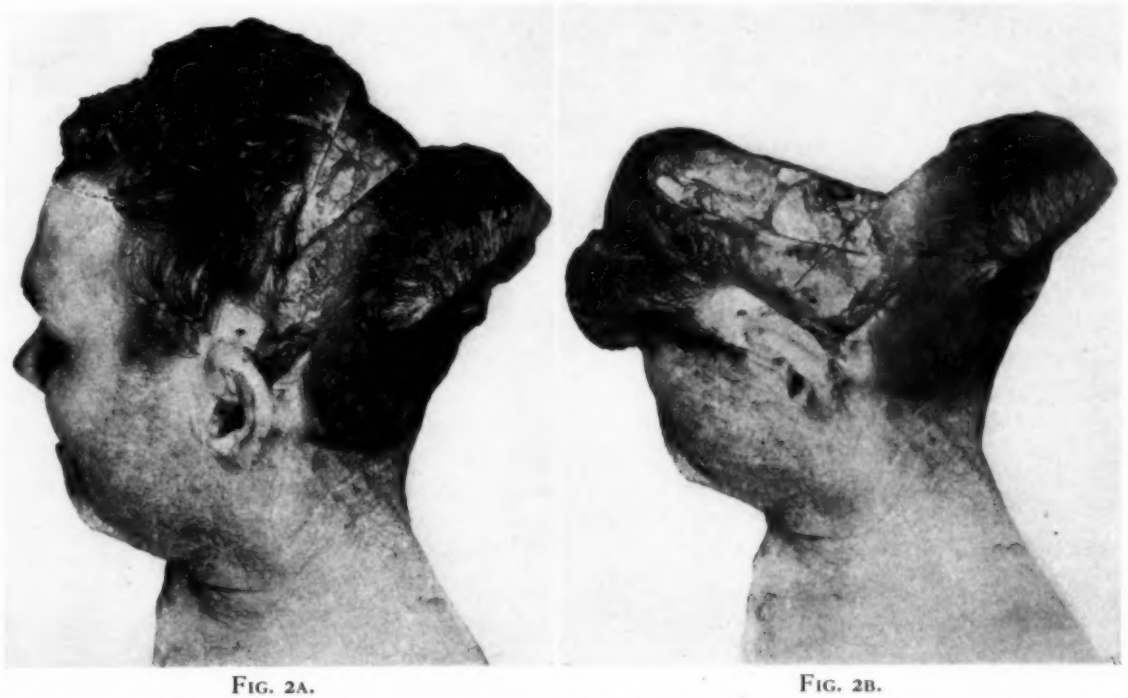

FIG. 2A. FIG. 2B.

FIG. 2. Case II. A. Small concealed penetrating stab wound through scalp above left ear. B. Scalp has been reflected and temporal muscle cut away to show small hole in skull. Tract passed through left temporosphenoidal lobe into left cerebral peduncle.

CASE II. Clinically undetected stab wound of the left side of head and brain. Homicidal assault with slender pointed weapon, probably an ice pick. Duration of illness five days.

*Clinical Record.* T. G., a sixty year old Italian man, was admitted to Bellevue Hospital on February 5, 1934. There was an indefinite history of assault three days before, at which time he was said to have received multiple contusions about the body and a laceration of the scalp. He had been found lying in bed, naked and semi-comatose. Further history was not obtainable.

On examination in the hospital, the patient was dehydrated, semi-comatose and incontinent of urine. The respirations were stertorous. The right pupil was slightly larger than the left, and the deep reflexes on the right side were more active than on the left. The Babinski and Hoffman reflexes were present and the abdominal reflex was absent on the right.

There was a right lower facial weakness. Numerous contusions and a few lacerations were noted on the thighs, elbows and scalp. Successive blood pressure readings were 158, 160, 130 mm. Hg systolic and 76, 90, 80 mm. diastolic, respectively. The spinal fluid was bloody. The temperature rose from 100° to 106°F., and the pulse rate from 80 to 135 per minute. The blood sugar was 363 mg. per cent; the urine contained 3 plus sugar and 2 plus acetone. The leucocytes numbered 17,000 with 72 per cent polymorphonuclears. The patient remained unconscious and died two days after admission.

Clinical diagnoses were: laceration of the left side of the brain; questionable fracture of the skull with extradural or subdural hemorrhage; multiple contusions of the body.

*Necropsy.* Anatomical Diagnosis: Penetrating stab wound of left temporal portion of scalp, left temporal muscle, squamous portion of left temporal bone, dura, left temporosphenoidal lobe, left cerebral peduncle; interpeduncular subarachnoid hemorrhage; multiple contusions about the body.

Fading ecchymoses were found on the thighs, right upper eyelid, right arm, left forearm and a few small abrasions on the knees and right elbow. On cutting away the hair from the left temporal region of the scalp, a small dry and crusted, transverse, slit-like stab wound, 3 mm. long was found 1.5 cm. above the left ear (Fig. 2 A). It was obscured by hair and its presence was not suspected until the skull had been opened and the brain removed at which time the stab wound in the brain was seen. The dura was then stripped from the inner surface of the skull and a 3 mm. circular perforation, filled with clotted blood, was observed in the squamous portion of the left temporal bone. The hole in the bone was not visible on the outer surface until the temporal muscle had been cut away (Fig. 2 B).

The tract of the stab wound passed medially from left to right slightly downwards and forwards and after penetrating the scalp, left temporal muscle and temporal bone, which was only 1.5 mm. in thickness at the point of perforation, continued through the dura and through the left temporo-sphenoidal lobe. There was an opening 3 mm. in diameter on one of the convolutions of the lateral surface of the lobe, the tract emerging through a 3 mm. slit on the medial and inferior surface adjacent to the left cerebral peduncle, which was also penetrated for a short distance through its basilar portion. The peduncular tissue in this region was contused and reddish gray in color. The length of the tract in the brain was 6.5 cm., and the entire length 7 cm. A small localized subarachnoid hemorrhage was found in the interpeduncular space. The vessels at the base were moderately sclerosed and a small intact aneurysm was found on the left posterior cerebral artery, a short distance from the circle of Willis.

Case III. Clinically undetected penetrating stab wound of the left side of the forehead skull and brain; intermeningeal hemorrhage; cerebral compression. Homicidal assault with slender pointed weapon, probably an ice pick. Duration of illness, six hours.

*Clinical Record.* G. S., a twenty-nine year old negro, was admitted to the Harlem Hospital on March 30, 1934, having been brought there by an unknown man who stated that the patient had been drinking heavily and had become involved in a fight.

The patient was alcoholic and deeply comatose. The respirations were stertorous. A small laceration was noted on the left side of the forehead; the right upper eyelid was ecchymotic, and the nose was bleeding. There was a flaccid paralysis of the left upper and lower extremities and of the right side of the face. The pupils were 4 mm. in diameter and did not react to light. The deep reflexes were decreased or absent on both sides. The pulse rate was 54 per minute and the rhythm irregular. The spinal fluid was bloody and under an increased pressure of 650 mm. The blood count revealed 12,200 leucocytes with 78 per cent polymorphonuclears; 4,310,000 erythrocytes with 75 per cent Hb. The patient remained in coma and died six hours after admission.

The clinical diagnosis was intracranial injury of unknown type.

*Necropsy.* Anatomical Diagnosis: Penetrating stab wound of left side of forehead, scalp, calvarium, dura, brain, left lateral sinus; left-sided subdural hemorrhage with right-sided cerebral compression; superficial stab wound of upper lip, abrasion and contusion of right side of face and cheek; ecchymosis of right upper eyelid; aspirated blood in lungs, swallowed blood in stomach.

Blood was found in the nose and mouth. The right upper eyelid was ecchymotic and a hematoma and superficial abrasion of the

outer end of the right eyebrow was seen. A small perforating stab wound 4 mm. in length was found on the left side of the forehead

ward from the wound of entrance in the left frontal lobe as far as the temporal lobe. A large amount of blood was found in the lateral

Fig. 3A. Fig. 3B.

Fig. 3. Case III. A. Small penetrating stab wound on left side of forehead mistaken for a laceration. B. With scalp reflected, corresponding hole in underlying frontal bone is visible. Tract extended backwards through left side of brain.

5 cm. to the left of the midline and 5 cm. above the supraorbital ridge (Fig. 3 A). The edges of the wound were sharp. The underlying frontal bone was also perforated by a small hole which on the outer table was triangular in shape, beveled anteriorly and undermined posteriorly (Fig. 3 B). The frontal bone measured 4 mm. in thickness. The tract of the stab wound passed backwards, downwards and medially, through the dura, the left frontal lobe, the left cerebral hemisphere, and then pierced the left lateral dural sinus, emerging on the posterior surface of the left occipital lobe, just above the tentorium, where it terminated. It measured 15 cm. in length. 60 c.c. of fluid and clotted blood were found in the left subdural space. The convolutions over the left cerebral hemisphere were molded; those over the right were flattened. A layer of subarachnoid hemorrhage extended backventricles, more on the left side, and in the third and fourth ventricles. On section, considerable hemorrhage was found along the entire tract.

Case IV. Clinically undetected knife wound of left side of scalp, calvarium and brain. Delayed secondary subcortical hemorrhage. Homicidal assault, probably with a thin-bladed knife. Duration of illness, thirteen days.

*Clinical Record.* W. B., a thirty-five year old negro, was admitted to the Harlem Hospital January 25, 1933, with a history of having been severely beaten about the head during an altercation on the morning of admission. He had not lost consciousness. A friend, who noticed that the patient was unable to speak and had a right-side paralysis, brought him to the hospital.

When first examined, he was conscious, not acutely ill, and exhibited a motor speech aphasia. The left parietal region of the scalp was swollen and very tender, and the nose was bleeding. During the examination, the patient cooperated well. He was able to utter sounds but not words or sentences, and he performed movements promptly. There was a motor weakness of the right arm and right lower facial muscles and a loss of sensation on the entire right side of the body. The right lower extremity was moved freely. The right knee jerk was more active than the left and the abdominal reflexes were diminished. The right pupil was 4 mm. in diameter and sluggish to light. The left pupil 2.5 mm., reacting promptly to light. The spinal fluid was under an increased pressure of over 300 mm. and was faintly blood-tinged. A subsequent neurological examination reported deep reflexes normal and equal, the right abdominal reflex absent and the pupils equal, reactive, and the fundi normal. The next day, his condition was unchanged. The blood pressure was 116 mm. Hg systolic and 68 mm. diastolic; the pulse was 61 per minute and the rhythm irregular. During the next few days the patient's condition improved with considerable restoration of motion in the right arm but with no power in the hand. Speech improved slightly but the hemianesthesia persisted. The pulse was 56 per minute and the rhythm regular. The spinal fluid was again blood-tinged and under a pressure of 220 mm. For several days his condition remained unchanged. *X*-rays of the skull were reported at first as suggestive of a fracture in the left parietal bone, but were subsequently reported negative. The temperature varied between 100° and 102°F.; blood count: 9200 leucocytes with 76 per cent polymorphonuclears; 4,100,000 erythrocytes, 75 per cent Hb.

On the thirteenth day after admission, as the patient was about to sit up in bed, he suddenly lapsed into coma, developed spasticity of all the extremities, and died in a few minutes.

The clinical diagnosis was fractured skull with possible left-sided epidural hemorrhage.

*Necropsy.* Anatomical Diagnosis: penetrating stab wound of the left parietal portion of the scalp, left parietal bone, dura, left parietal cerebral cortex, localized subarachnoid hemorrhage, secondary subcortical cerebral hemorrhage, cerebral compression.

The hair on the scalp was of the short, thick, kinky, black variety. A healed scar 1.5 cm. long was found in the left parietal region, its anterior end 10 cm. above and 2 cm. in front of the left auditory meatus. When the scalp was reflected, ecchymosis was visible around the scar. Beneath it a thin linear stab wound, 1.3 cm. in length, was found penetrating the left parietal bone (Fig. 4 A). In this region the bone was only 3 mm. in thickness. The calvarium was not very hard. There was a corresponding perforation in the dura on the left side, 1 cm. in length, and beneath it a small localized subdural hemorrhage. The underlying cerebrum in the region of the fissure of Rolando was softened and covered by a subarachnoid hemorrhage 5 cm. in diameter. The stab wound penetrated the brain through the precentral gyrus, extending through the fissure into the postcentral gyrus for a distance of 2 cm. The motor area involved was that of the arm and head. The left cerebral hemisphere was swollen and larger than the right and the convolutions over both were flattened. After the brain was hardened in formalin, a horizontal section was made 1 cm. above the level of the stab wound exposing a large fresh subcortical hemorrhage 5 cm. in diameter extending posteriorly and medially from the end of the wound into the white matter of the left parietal lobe (Fig. 4 B).

Case V. Stab wound of left side of neck and left common carotid artery. Mural thrombosis at site of stab wound. Embolism to left middle cerebral artery. Homicidal assault, probably with a pocket knife. Duration of illness thirty-six hours.

*Clinical Record.* O. F. G., a twenty-two year old negro, was brought to the Harlem Hospital by two men on November 20, 1932. He had been stabbed in the neck and had bled considerably before admission.

The patient was unconscious and in shock. The respirations were 24 per minute; the pulse rate 112, irregular and of poor quality. There was a bleeding stab wound 1.5 cm. long, situated 2 cm. above the middle of the left clavicle. The conjunctivae and mucous membranes were pale. The blood pressure was 60 mm. Hg systolic and 40 mm. Hg diastolic. Under ether anesthesia, the wound was enlarged and explored and then packed with gauze. An infusion of 1000 c.c. of gum acacia was given. The blood pressure rose to 130 mm.

Hg systolic and 92 mm. Hg diastolic. The pulse rate became slower and the rhythm regular. The wound continued to bleed and was re-

In the left common carotid artery, 4 cm. above its origin from the aorta, an oval shaped knife wound, 7 mm. vertically and 3 mm.

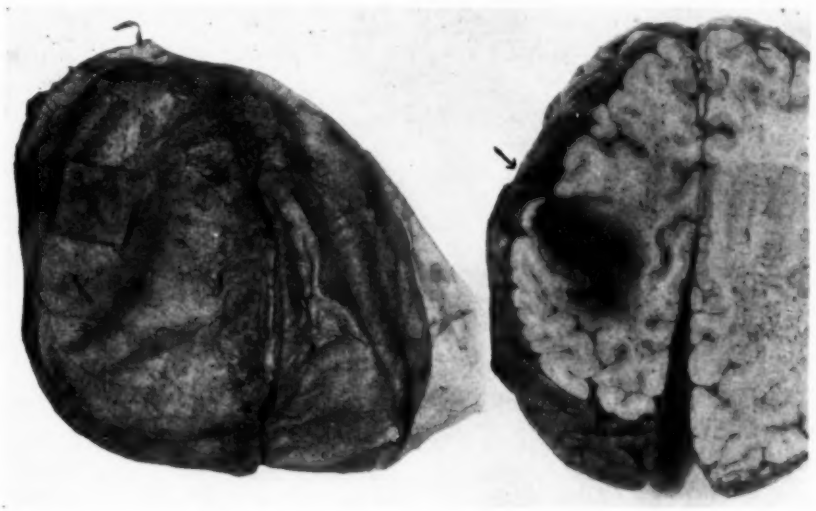

Fig. 4A. Fig. 4B.

Fig. 4. Case IV. A. Thin knife wound through left parietal bone and underlying dura. Upper surface of dura is shown with a small square of bone containing stab wound placed above corresponding slit in dura. B. Entrance of stab wound on surface of left cerebral hemisphere just anterior to fissure of Rolando is shown by arrow. Large secondary subcortical hemorrhage is visible.

packed. Consciousness had returned. The following morning the patient suddenly became deeply comatose; the respiration stertorous. The pupils were dilated and fixed to light, and the right eyeball rotated externally. There was a spastic paralysis of the right upper extremity and the abdominal reflexes were absent. The pulse became rapid and weak and the blood pressure fell sharply. The spinal fluid was clear. The patient died shortly after coma set in.

The clinical diagnosis was penetrating stab wound of the neck with possible large vessel injury. A cerebral embolism was suspected.

*Necropsy*. Anatomical Diagnosis: Stab wound of left side of neck, left common carotid artery, left side of trachea. Hemorrhage into carotid sheath, neck, superior and posterior mediastinum. Mural thrombosis at site of stab wound in carotid artery. Embolism to left middle cerebral artery. Cerebral infarction and compression.

The tract of the stab wound on the left side of the neck was directed downwards, medially and backwards, and measured approximately 6 cm. in length. There was extensive hemorrhage in the tissues of the neck, left carotid sheath and superior mediastinum. transversely, was found in the posterior wall. This wound was plugged by a soft antemortem thrombus which protruded into the lumen of the vessel (Fig. 5 B). The tract of the stab wound had passed medially to the jugular vein, cutting the posterior surface of the carotid artery and continuing through the left lateral wall of the trachea, which was not completely penetrated. There was a large amount of hemorrhage between the trachea and the esophagus which extended down into the posterior mediastinum.

On removing the calvarium and dura, the convolutions over the entire cerebrum were flattened. The left hemisphere was swollen and slightly softened. The longitudinal fissure was displaced to the right. With the exception of a small portion of the frontal and occipital lobes, the surface of the left hemisphere was pinkish in color. This congested area extended down over the left temporo-sphenoidal lobe. The left middle cerebral artery was completely occluded by an embolus (Fig. 5 A). On horizontal section through the brain, there was a distinct swelling and softening of the basal ganglia and that portion of the cerebrum supplied by the middle cerebral artery. The

process was very recent and the landmarks were not destroyed.

Case VI. Stab wound of the left side of neck, left common carotid artery and small tributaries of jugular vein; mural thrombosis of left common carotid artery at site of wound; cerebral and pulmonary embolism and infarction. Homicidal assault, probably with a knife. Duration of illness two days.

Fig. 5A. Fig. 5B.

Fig. 5. Case V. Stab wound of neck and left common carotid artery with thrombosis and cerebral embolism. A. Left middle cerebral artery is occluded by an embolus. Artery on right is collapsed and empty. View from under surface, horizontal section of brain exposing fissures of Sylvius and vessels. B. Left common carotid artery opened to show stab wound plugged with thrombus which protrudes into lumen.

*Clinical Record.* J. C., an eighteen year old negro, was stabbed in the neck and left side of the head during an altercation. Following this there was considerable hemorrhage and the patient lost consciousness, which he regained after admission to the Harlem Hospital on February 21, 1933.

On examination, the respirations were labored, the mucous membranes and conjunctivae were pale; the pulse regular and of good volume. There was a deep laceration of the scalp behind the left ear which penetrated to the outer table of the skull. There was also a stab wound 1.5 cm. in length, in the posterior triangle of the left side of the neck, 5 cm. above the middle of the clavicle. There was a large hematoma in the anterior portion of the neck. The wound was not bleeding externally. The following morning a right-sided facial paralysis and convulsive movements of the right upper extremity and neck suddenly developed, and the patient became unconscious. On the right side the abdominal reflex was absent and a Babinski was present, and on the left the biceps and patellar reflexes were more active. The spinal fluid was clear. The scalp and neck wounds were explored surgically and the former sutured and the latter packed. The patient was given a transfusion of 300 c.c. of blood. The blood pressure, which on admission was 70 mm. Hg systolic and 48 mm. diastolic, rose to 125 mm. systolic and 80 mm. diastolic, subsequently falling to 80 mm. systolic, 40 mm. diastolic. The temperature rose from 98° to 104°F., and the pulse rate from 100 to 150 per minute during the course of his illness. Blood count was: 3,100,000 erythrocytes, with 65 per cent Hb.; 8800 leucocytes, 70 per cent polymorphonuclears. The day following the onset of the paralysis the patient died.

The clinical diagnosis was stab wound of the neck involving the great vessels, cerebral embolism with right-sided hemiplegia. Laceration of scalp. The possibility of a penetrating wound of the skull beneath the scalp laceration was also considered.

*Necropsy:* Anatomical Diagnosis: Incised wound of left side of scalp and outer table of skull. Stab wound of left side of neck, left common carotid artery and smaller tributaries of jugular vein; thrombosis of carotid artery at site of stab wound; hemorrhage into tissues of neck; embolism to left middle cerebral artery, cerebral infarction and compression; pulmonary infarction; bronchopneumonia.

The stab wound on the left side of the neck had been enlarged surgically. There was considerable hemorrhage in the fascial planes of the neck and in the left carotid sheath, which extended downward in the posterior mediastinum almost to the diaphragm. The jugular vein was not injured but several ligated veins were seen in the wound. A stab wound was found in the posterior wall of the left common carotid artery 5 cm. below its bifurcation. The wound which was 1.3 cm. long was plugged with a soft friable antemortem thrombus which projected into the lumen of the artery. The tract of the stab wound was 4 cm. in length. Extensive areas of hemorrhagic infarction and small areas of bronchopneumonia were found in the lungs.

The lacerated wound of the scalp was 4 inches in length and extended down to the outer table of the skull. The calvarium was thick. The convolutions on the surface of the brain were flattened and the blood vessels were congested over that portion of the left cerebral hemisphere supplied by the middle cerebral artery. A small embolus was found plugging the lumen of the left middle cerebral artery about 2 cm. from its origin. On section, the left basal ganglia and left cerebral hemisphere, with the exception of the anterior portion of the frontal lobe and posterior portion of the occipital lobe, were swollen and softened but the markings were not destroyed. The appearance was that of a fresh infarction.

Case vii. Stab wound of neck, left innominate vein and arch of aorta with formation of an arteriovenous aneurysm dissecting into superior and anterior mediastinum and rupturing into left pleural cavity. Massive left hemothorax. Homicidal assault with a knife. Duration of illness, thirteen days.

*Clinical Record.* A W., a twenty-four year old negro, was admitted in coma to the Harlem Hospital on May 6, 1934, after having been stabbed in the lower part of the neck.

On examination, the patient was acutely alcoholic and semi-stuporous. A vertical stab wound, 1.5 cm. long, was seen 1.5 cm. to the left of the midline with its lower end at the level of the suprasternal notch. The wound bled slightly and was packed with gauze. There was no subcutaneous emphysema or hematoma. Several days after admission, it was noticed that the heart was displaced to the right, and that the right radial pulse was stronger than the left. A bruit lasting throughout the entire cardiac cycle was heard over the precordium and was very loud over the second left interspace and over the left side of the neck. The left side of the chest was tympanitic below the clavicle and dullness, diminished breathing and bronchophony were noticed over the base. An *x*-ray picture revealed a large collection of fluid and air in the left pleural cavity. On the twelfth day, the patient was in collapse. The blood pressure which was 120 mm. Hg systolic, 70 mm. diastolic, on admission, was recorded as 90 mm. systolic and 40 mm. diastolic. The pulse was more rapid. The entire left chest was very flat on percussion, and the heart was more displaced to the right. The bruit was diminished in intensity. The following day the patient died.

The clinical diagnosis was a stab wound of the neck and chest, with injury to the large blood vessels; traumatic arteriovenous aneurysm with secondary hemorrhage into the left pleural cavity.

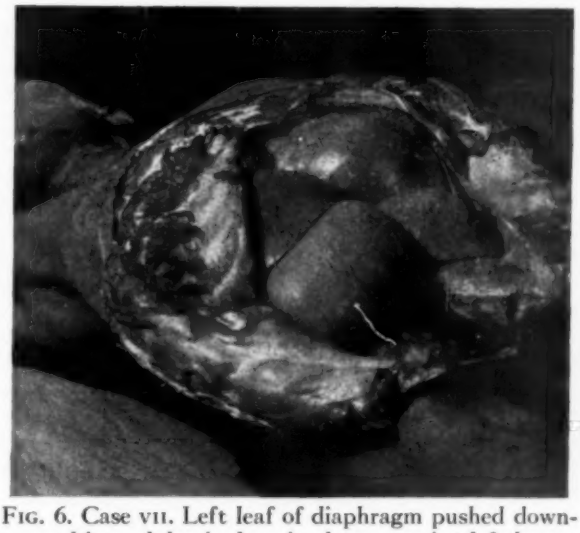

Fig. 6. Case vii. Left leaf of diaphragm pushed downward into abdominal cavity by a massive left hemothorax resulting from slow leakage into left pleural cavity of a ruptured dissecting arteriovenous aneurysm which developed after a stab wound of neck, left innominate vein and aorta. Bulging diaphragm displaced spleen, liver and stomach downward and to right.

*Necropsy.* Anatomical Diagnosis: Stab wound of lower anterior region of neck, left innominate vein and aorta. Arteriovenous aneurysm with aneurysmal dissection of the anterior mediastinum and rupture into the left pleural cavity. Massive left-sided hemothorax. Compression atelectasis of left lung and depression of left dome of diaphragm. Anemia of organs. Contracted heart.

The stab wound on the neck above the suprasternal notch was covered with a dry brown crust. The tract was directed downwards and backwards and measured 6.5 cm. in length. After piercing the muscles of the neck, it transfixed the left innominate vein and then passed through the arch of the aorta medial to the origin of the left subclavian artery, then continuing across the lumen of the aorta and terminating in a vertical cut on its posterior wall just below the isthmus. Both wounds in the innominate vein and the wound of entrance in the aorta were 1.3 cm. in length. The cut on the posterior wall of the aorta was 2 cm. in length. A considerable amount of extravasated blood was found between the

aorta and the left innominate vein extending up into the neck and into the superior mediastinum. The stab wound in the anterior wall of the innominate vein communicated with a large extravasation of fluid and clotted blood which extended downward as a false aneurysmal sac into the tissues of the anterior mediastinum as far as the diaphragm. This sac in front of the upper part of the pericardium, when sectioned, measured 4 cm. in thickness. Along the left margin of this sac, a rupture was found 2 cm. in length, communicating with the left pleural cavity. The left pleural cavity was distended with more than 4200 c.c. of reddish brown sanguineous fluid and dark red clotted blood. The left lung was completely collapsed and compressed against the vertebral column. The heart was displaced to the right and the left dome of the diaphragm was pushed down, its under surface bulging downward into the abdominal cavity and displacing the abdominal viscera downward and to the right (Fig. 6). The chambers of the heart were contracted and the liver, spleen and kidneys were anemic.

## COMMENT

From a consideration of the first four cases described, it is apparent that a forceful blow with a sharp pointed weapon may readily penetrate the skull and brain and produce only a small surface wound which may escape detection completely or be mistaken for a superficial lesion. In Case I, a correct diagnosis of a fatal stab wound was made. In Cases II and IV, the fatal scalp wounds were concealed by the hair and not detected. In Case III, the small fatal wound on the forehead was mistaken for a laceration. In these three instances, traumatic intracranial lesions were anticipated because of the history of assault and clinical findings. At necropsy, the concealed wounds were not discovered until after the calvarium had been removed in Case II and the scalp reflected in Case IV.

Penetration by a pointed weapon can readily occur through the thin squamous portion of the temporal bone as in Cases I and II, or through a thin frontal or parietal bone, as in Cases III and IV, respectively. Thin calvaria are frequently observed in many individuals and in all races of people. The degree of hardness or compactness of the skull, even though it is of normal or increased thickness, is also an important factor in its resistance to penetration by a pointed weapon. Occasionally, at necropsy, a piece of a knife blade or ice pick is found embedded in the deeper layers of the scalp or in the outer table of the skull, having presumably been forcibly broken off on striking the surface of an impenetrable compact hard skull. The writer has seen such examples.

In the absence of other signs of violence and without a history of assault, with clinical signs consistent with disease as well as with trauma, the possibility of not detecting a concealed wound of the type described, is very great. In such a case, death may be attributed to natural causes and unless the circumstances of the case are suspicious, or permission for necropsy obtained, a postmortem examination may not be made and a serious crime pass undetected.

The duration of the illness in the first four cases is of interest. In Cases I and II, in which the patients lived five days, there was only a slight amount of hemorrhage along the slender contused tracts in the brain, and death evidently was caused by the resultant cerebral edema and compression. In Case III, death occurred rapidly in six hours, and was undoubtedly hastened by the severe cerebral injury, the traumatic cerebral hemorrhage, and also by the subdural hemorrhage resulting from the wound in the lateral dural sinus. In Case IV, the patient lived thirteen days. He was stabbed through the brain in the vicinity of the left fissure of Rolando, and the motor arm, face and speech areas and the adjacent sensory area were injured. He did not lose consciousness until the onset of a large secondary subcortical hemorrhage beneath the site of the stab wound. This hemor-

rhage apparently occurred shortly before death.

Cases V and VI are similar examples of stab wounds of the left side of the neck. Although the surface wounds were small, the profuse hemorrhage suggested the possibility of damage to the deep vessels. In each case, a stab wound of the left common carotid artery was plugged by the formation of a thrombus, and death resulted not from local hemorrhage, but from embolism to the left middle cerebral artery, resulting in extensive cerebral infarction. In Case VI, the presence of an additional deep incised wound of the scalp, which reached the calvarium, suggested the possibility that the neurological signs which had suddenly developed might have resulted from a penetrating skull wound. In this case, there was also considerable pulmonary infarction, probably secondary to the thromboses in the smaller injured veins of the neck.

Case VII represents a very remarkable example of a stab wound of the neck which penetrated the superior mediastinum, passing through the left innominate vein and the arch of the aorta. An arteriovenous aneurysm was established between these vessels and a hemorrhage spreading from the venous side of the aneurysm produced an unusual dissection of the tissues of the anterior mediastinum, converting it into an aneurysmal sac which subsequently slowly leaked into the left pleural cavity. A left-sided massive hemothorax, containing more than 4 liters of blood, accumulated. Death occurred thirteen days after the stabbing, a remarkably long duration of illness after an injury of this type. This case also illustrates how a very large volume of blood may accumulate in a serous cavity when hemorrhage occurs slowly.

## SUMMARY

Four examples of fatal penetrating stab wounds of the head and brain are described. In three of these, the weapon used was probably an ice pick, and in one, a thin-bladed knife. In two of these cases, the small surface wounds on the scalp were concealed by the hair and not detected clinically, and in one case, a small wound on the forehead was mistaken for a superficial laceration.

In two examples of stab wounds of the neck and left common carotid artery, a mural thrombus developed in the artery wound and gave rise to an embolus which lodged in the left middle cerebral artery, causing death from cerebral infarction.

A fatal stab wound of the neck and left innominate vein and arch of the aorta resulted in an arteriovenous aneurysm which dissected the anterior mediastinum and ruptured into the left pleural cavity where a massive hemothorax was formed.

I am indebted to Dr. Chas. S. B. Cassassa, Acting Surgical Director of the Harlem Hospital and to Dr. Carl Burdick, Director of the 4th Surgical Division of Bellevue Hospital, for permission to use the clinical records of the cases reported in this paper. The necropsies in Cases III, IV, VI and VII were performed by Dr. Thomas A. Gonzales who kindly consented to their inclusion in this report.

# INJURIES TO THORACIC DUCT

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IT is a comparatively rare occurrence for the thoracic duct to be injured because of its anatomical location. The traumatic surgeon, however, should be sufficiently acquainted with the phenomenon to recognize such a lesion when it occurs and be prepared to meet the emergency properly.

A penetrating wound just above the left sternoclavicular joint is the vulnerable spot for injury to reach the thoracic duct before it enters the left subclavian vein. A dagger, knife, or a piece of glass might cause it, as well as a bullet, but the greatest number of reported occurrences have been during cervical operations. From this region injury to the duct will cause drainage to issue externally. Injuries from other directions which reach the thoracic duct deeper in the chest will nearly always cause an effusion into the chest cavity and develop a chylous thorax.

Numerous studies have shown the anatomical position and affiliations of the thoracic duct to have wide variations. Normally it ascends the thorax in front of the bodies of the vertebral column. When reaching the level of the seventh cervical vertebra it crosses over to the left side between the azygos vein and the aorta, thence becoming more exposed anteriorly, up to the point of entry into the subclavian vein which is over the apex of the lung. It has collateral connections with the azygos vein, which are usually quite small, and frequently a second terminal branch.

Chyle is a milky white fluid, sometimes called "white blood," but which does not clot. It conveys to the blood all the fat absorbed from the intestinal lacteals, a vital function. Complete loss of this substance through drainage causes starvation and death, but the body may carry on during partial deprivation.

Recognition of an injury to the thoracic duct is comparatively easy. The presence of milk white fluid in the wound of the neck or thorax is a direct indication.

Treatment of wounds of the thoracic duct is quite unsatisfactory. Four methods have been suggested: (1) Suture repair of laceration in the duct. This is best but nearly always impossible. (2) Implantation into an adjoining vein, a difficult procedure but yielding good results if it can be accomplished. (3) Ligation of the duct. This is particularly good because nearly 50 per cent of the cases have a second terminal branch to carry on. Yet frequently the chyle leaks through the ligature the following day through failure to clot. (4) Tamponade. Most of the cases in the literature have been treated in this fashion, with fair results. A pressure pack of vaseline gauze serves perhaps as well as any.

Recovery from these injuries occurs in more than 50 per cent of the cases. The deciding factor is whether the body nutrition can be maintained. The ensuing course is usually a profuse discharge of chyle, which slowly diminishes and ceases in from two days to a month. Sometimes a permanent fistula results but the creation of back pressure nearly always causes a dilatation of the collateral connections to the azygos veins until they enlarge sufficiently to care for the full flow. It is felt that most of the successful cases spontaneously close no matter what treatment is applied.

From our survey of the literature we have collected only 8 cases due to non-surgical injury and resulting in external drainage, 17 traumatic cases mentioned by Lilienthal resulted in chylous thorax and one in chylous ascites.

Lilienthal lists 24 cases which occurred other than at operations.

| | |
|---|---|
| Gunshot | 4 |
| Stab wound | 2 |
| Fractured rib or clavicle | 6 |
| Fractured vertebrae | 2 |
| Fractured ribs and vertebrae | 3 |
| Contrecoup | 7 |

Twelve of these patients died and twelve recovered. The cause of death is given as compression of the lungs and starvation.

Sajous and others have reported groups of cases resulting from accidental injury during operation. One author lists forty-one. In general these involve similar problems.

## CASE REPORT

A. D., a boy, aged thirteen, was brought to the Hackensack Hospital by ambulance about 10 o'clock at night. He was struck by an automobile while riding a bicycle. The window by the driver's seat was shattered and the boy's arm went through the glass.

He recovered consciousness on admission to the hospital but was suffering from severe shock. Bleeding from a long laceration on the left side of his neck was easily checked by pressure bandage. Weakness of the left arm was noted. There were minor lacerations. That night he was treated for shock and fracture of the skull.

The following morning his general condition had improved. It was noted that his left arm was almost completely paralysed. He was taken to the operating room for repair of the laceration of the neck which extended 4 inches from the margin of the cricoid cartilage just to the left of the midline diagonally to the lateral border of the neck. At first glance it seemed superficial. The platysma was cut and also the medial head of the sternocleidomastoid muscle. The skin margins gaped 2½ inches apart. We then became aware of a milky white chyle and the wound was explored. Several layers of tissue were separated but the vessel source could not be found. Evidently a long splinter of glass had pierced the thoracic duct without injuring the surrounding vessels. Two purse-string ligatures were passed as deeply as possible and the flow was diminished to almost nil. Severed nerves were searched for in vain. The cut did not extend as far back as the brachial plexus. Vaseline gauze was packed in tightly. Four silkworm sutures were placed but not tied.

Twenty-four hours later the wound was clean but was pouring out chyle profusely. The sutures were pulled together for secondary approximation and to stop the flow of chyle. The following day with the drainage stopped his temperature shot to 104°F. The wound was opened, a free flow of chyle reestablished and the temperature dropped. The flow became freer. Dressings and bedclothes were constantly saturated with chyle, to an estimated amount of from one to two quarts daily.

This continued for three weeks. Drinking would immediately start up the flow but methylene blue given by mouth did not show through. His general condition remained fair and no digestive disturbance was noted. He gained 2 pounds before final closure.

The urine was negative. Hemoglobin 70 per cent, R.B.C. 3,900,000, W.B.C. 10,100, polymorphonuclears 78 per cent. A 2 per cent solution of potassium iodide was given by mouth but the chyle did not contain iodine. The secretion was compared with milk. Babcock fat determination 4.5 per cent and Benedict's solution was reduced. Microscopically it was dissimilar to milk. Renin + HCl + specimen gave a scant precipitate.

The wound slowly granulated in, appearing healthy throughout and tamponade repeated again after three weeks effectually stopped the flow. Subsequent recovery was complete except for a brachial plexus lesion which was later successfully operated upon at the Neurological Institute.

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# TRAUMA AND MALIGNANT TUMORS

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THE question of how far a single mechanical trauma may be assumed to be the sole etiological agent in the production of a malignant tumor is receiving more and more attention of late with the extension of labor compensation laws and the increased number of accidents following the widespread use of motor vehicles. Speaking generally, the opinion of almost all pathologists and experimental students of cancer is that a single trauma cannot cause a malignant neoplasm. On the other hand a good many clinicians give court testimony or publish articles supporting the thesis that in rare instances a single trauma does cause the appearance of a malignant neoplasm. This correlation is assumed chiefly in connection with the sarcomata of bone or soft tissues; it is rarely held for the carcinomata. The courts on the whole take a neutral position and juries frequently find for the plaintiff if the evidence is contradictory. The feeling in compensation cases is often that since the doctors disagree, the plaintiff should have a small award inasmuch as the disease is fatal, even if an absolutely clear-cut relationship between the injury and the appearance of the tumor cannot be established.

The reasons for the differences in opinion between the laboratory workers and the clinicians are mainly as follows:

Extensive experimental work on animals has been done by inflicting various types of physical tissue injuries without any recorded instance of the production of a malignant tumor. Yet cancer is easily produced in these same animals by the action of chemicals, chronic inflammation due to irritants, physical agents such as radium[1] or *x*-ray and parasites, all of which produce cancer in man with equal facility, though of course in the case of the parasites the types effective in the human being differ from those which are found to be carcinogenic in animals. The pathologist cannot understand why if it is so easy to produce cancer in animals by the same agents that act effectively in human beings, is it impossible to produce cancer by a trauma in these animals if trauma is a factor in human neoplasms. He is also daily confronted with the discovery of undiagnosed cancer in persons coming to autopsy and, therefore is, always thinking of a tumor unsuspected by the patient and undiagnosed by the physician which may be revealed by a trauma. When from 10 to 20 per cent of undiagnosed cancer cases come to the autopsy table there is always in his mind the possibility of a coincidental occurrence of a neoplasm at the time of the injury. Further he is familiar with the statistical reports which show that there has been no obvious increase in cancer following injuries during the great war and that certain countries with extremely accurate death certification, despite the increase in industrial and motor accidents, show no increase in neoplastic disease in their official records.

The clinician on the other hand sees the dramatic aspect of the situation. A patient states very definitely that though in perfect health a tumor has appeared after an injury at the site of a blow so the gravity of the situation makes a strong impression upon the physician.

It is too frequently assumed that almost anyone without much experience can diagnose a tumor from a microscopic section or that microscopic examination may be unimportant as compared to clinical judgment. Unfortunately this is not true, as any pathologist with a large consulting practice knows, for it is some-

times only with the greatest difficulty that a differential diagnosis can be made between certain types of traumatic bone repair and a malignant neoplasm. The experienced pathologist is also aware of the fact that while clinical experience is in many cases very capable of affording an accurate diagnosis of a malignant growth, it not infrequently fails, especially with the sarcomata. It is of the greatest importance therefore that all these alleged cases of traumatic production of cancer be studied thoroughly, and some real progress in this branch of pathology may be expected if large institutions which are equipped to make and keep careful records, clinical, radiological and pathological, study such material and make it available for the guidance of the experts who are called upon to give testimony in court. In this way cases may be reviewed from time to time in the light of the changing knowledge of neoplasms and form homogeneous groups of tumors of special types in which at least some of the circumstances surrounding the early stages of a tumor and the position which the trauma may have held in that history are clearly defined. For instance, it is recorded in Troell's excellent paper, referred to later, that a careful gross and microscopic study was made of a number of bone sarcomata considered as traumatic in origin from the history but that no evidence of injury was found at the site of the tumor; hence the cases had to be excluded from the trauma group.

If each hospital preserved an adequate museum to which such tumors might be admitted with their full histories, and a critical study of the traumatic factor in each one was carefully appended, it might be that some useful information could be collected. This has frequently been done, especially in the case of the more superficial tumors where the presence of scars from injury or burns has offered indisputable evidence of the importance of trauma as a sensitizing agent for the ultimate appearance of cancer, though usually, as is discussed later, a period of years intervenes between the development of the neoplasm and the injury to the scar, and a much longer period from the time of the original injury and the secondary one; but a large number of other tumors, especially in the sarcoma group, may offer no physical indication as to their past history, and therefore our knowledge of such a relationship remains largely speculative.

An admirable illustration of the possibilities of confusion which may waylay the unwary is contained in the following recent report by Barret:[2]

> A man of forty-five fell and fractured the left femur. *X*-ray pictures taken at that time were interpreted as showing nothing but a fracture. Healing was rather slow but after about three months the patient got about. Eight months later he had a pathological fracture at the site of the previous injury. The *x*-ray showed a voluminous tumor diagnosed clinically as an osteosarcoma and the leg was amputated. Microscopic examination, however, showed that instead of a sarcoma the tumor was an alveolar growth which Oberling regarded as probably originating from the thyroid gland although no evidence of a neoplasm could be found on clinical examination of that organ.

In the discussion of this paper Barret refers to two other examples published by Balthazard:[3]

> The first was that of a robust man of fifty-four who slipped while carrying a heavy load and was found to have a subtrochanteric fracture of the femur. At the time it was reported that the radiographic examination revealed nothing abnormal except the fracture, but two months later the physician representing the insurance company noted that a tumor appeared at the site of the fracture and three and one-half months after the accident the patient died. The clinical diagnosis was obviously a traumatic osteosarcoma, but on careful resurvey of the original radiograph evidences of bone destruction were found and the later radiograph showed extensive involvement of the shaft of the femur, so that it was evident that either a primary myeloma or a metastatic

deposit from a carcinoma whose existence had not been recognized was present before the injury. Unfortunately a full record of the material is not available as no autopsy was done.

In the second case cited a laborer of twenty-three years was shot in the left knee. The wound healed rapidly and the patient left the hospital in a short time. Later he complained of pain in the calf of the leg and the internal condyle was found to be rarefied; there was a shadow in the upper portion of the popliteal space. This shadow was thought to be an abscess but no evidence of infection was found. Before the microscopic examination of this material was completed there was such a rapid increase in the popliteal tumor that the leg was amputated. When the report on the biopsy was obtained it recorded only inflammatory tissue and detailed examination of the suspected area from the amputated limb, while it showed considerable bone production with giant cells, was in the opinion of Professor Roussy only an inflammatory lesion.

These three interesting medicolegal cases illustrate only too clearly the necessity for full information before an opinion is rendered.

The legal decisions which have been rendered in these and similar cases can of course add nothing to the facts. They merely indicate the trend of social and economic thought with regard to compensation for persons who develop tumors while employed in industry, or who have suffered accidents of various sorts. Since a legal decision is made by lay persons, it may have little to do with the facts of medicine, and legal opinions, while they establish precedent, do not contribute to medical knowledge.

There is apparent in the literature of clinical medicine a strong desire to explain the phenomena which bring about disaster, and even when no logical explanation is obvious, the habit of medical philosophy appears to enter in and permit the assumption of some mysterious outside factors. This is perhaps derived from the German literature, which has formed so large a part of the scientific background of medicine, but one cannot overlook the element of the supernatural which causes the German clinician, having eliminated all reasonable possibility, to assume that trauma is the *auslösende Moment*, or the immediately exciting cause which has to be invoked if the transition from a normal to a malignant tissue is to be adequately explained. This explanation is to be found in so many of the otherwise clear and logical discussions that its presence alone excites scepticism. The confidence in predisposition occupies the same place in much of the contemporary literature. Yet any valid evidence for such a vague idea as predisposition is not at present available. The mere occurrence of high cancer rates in one family or one strain of animals and low rates in another does not prove immunity or resistance. (See on this point Curtis, Dunning and Bullock.[4])

A sharp distinction must be drawn between the effect of a single mechanical injury and the long-continued irritations which may result from an injury which has been the origin of some scar, ulcer, or special inflammatory lesion upon which a tumor later develops. In the latter group, there are unquestionably many genuine cases, and even hitherto unsuspected agents are being discovered which will stand the scrutiny alike of the statistician and the physician. Skin carcinoma, while ordinarily easily recognized microscopically, may not always be correctly diagnosed by those unfamiliar with healing of injuries or chronic inflammatory lesions, various dermatoses and benign lesions. Scars, rarely, even in childhood, may be the site of epithelioma, as in the case reported by DuBois[5] in which a child of ten years sustained a lacerated wound of the forehead and five months later a basal-cell epithelioma appeared which was microscopically diagnosed. Most of the cases, however, are in adults in whom irritation has been present for a considerable length of time, as in the case reported by Maiditsch[6] of a man sixty-five years of age who had at the age of

fteen received a lacerated wound of the eg which ulcerated fifty-one years later nd formed the basis for an extensive pithelioma. The average elapsed period or the appearance of the malignant lesion s about thirty-two years. (See for examples Arndt,[7] also Treves and Pack.[8])

This group of tumors has also varied in position, being found on the face and extremities in about equal numbers. Burns with asphalt,[9] tar, fat, acids, hot metals, various fluids, and excessive exposure to sunlight, and many types of lacerated wounds have been described as the basis of scars which have ulcerated, either spontaneously or following a secondary trauma, and then a neoplasm has developed in the site of injury. Maude Slye[10] has recorded a number of neoplasms arising in mice at the site of healing injuries or the resultant scars. Special types of irritants, such as ulceration with a pessary, of which 3 new cases, in addition to 19 or more in the German literature, have been reported by von Wolff[11] which when examined eleven and thirty years after their insertion were found to have caused epithelioma. In von Wolff's opinion irritation, infection, and predisposition were all necessary for the production of such a tumor. But in none of these examples can a single trauma be incriminated.

A tumor of the tongue is reported by Ullmann[12] in a man aged forty whose tongue was traumatized for only "a short time" by a filled tooth. But the whole value of the report is vitiated by the fact that the patient's story of feeling a slight pain in the tongue a few days before is accepted as fixing the date of origin of the neoplasm. The pain was undoubtedly due to the epithelioma already present.

If the accuracy of such reports is accepted as absolute, it is obvious that the interval necessary for the appearance of the tumor cannot with certainty be predicted either from the age of the patient, the duration of the irritant, or from the type of original injury. On the other hand, when the most accurate records are studied, the length of time which must elapse between the beginning of the irritation process and the appearance of the neoplasm is always a matter of years. The best examples are the tar, oil and *x*-ray cancers in which the period of exposure is accurately known. Even with the highly carcinogenic substances recently discovered, of which methylcholanthrene is the most active, at least two and one-half months must elapse before a carcinoma appears in a mouse, or a tenth of the animal's life.

It is noteworthy that cancer of the breast has recently received scant attention from the advocates of trauma as an etiological factor in cancer. However, there is one report of a tumor in a man forty-eight years of age who had received a war wound of the right nipple fourteen years before.[13] A claim is also made[14] that a trauma to the breast caused a rapid spread of a breast carcinoma which was undoubtedly present before the accident. Such traumatic spread is generally accepted as possible.

Fischer-Wasels, who has vigorously opposed the theory that brain tumors are to any considerable extent caused by trauma, admits the possibility that growth of a carcinoma of the lung might be somewhat accelerated by a trauma to the chest.[15]

The brain tumors have during recent years perhaps aroused more interest and discussion than almost any other group, the extreme position being taken by R. Beneke,[16] who believes that at least 40 per cent of brain tumors are caused by trauma, and includes under this term not only falls and blows on the head causing violent injuries and unconsciousness down to those not even causing headache, but also fright or shock without physical injury; also accidents causing injury to other parts of the body; or anything else capable of setting up direct or reflex ischemia of the brain and resulting necrosis of glial tissue, might be

the cause of a glioma. This position is definitely opposed by Fischer-Wasels, who does not believe that a transitory vasospasm can be the excitant for tumors, necrosis, or other pathological changes, and who insists upon a scientific study of the data rather than the attachment of too much importance to the possible connection in any one patient between an intracranial tumor and trauma to the head. Nevertheless, in Fischer-Wasels' case a bullet wound of the brain occurring nine years before is assumed by him to be the cause of a glioma which apparently arose in the scar tissue.

Parker and Kernohan[17] made a very complete study of a large number of cerebral cases in an effort to eliminate the factor of coincidence, and report upon careful histories taken to evaluate trauma in 431 cases of glioma. Injury was stated to have been present in 58 cases, or 13.4 per cent. In the control cases without tumors injury was claimed in 45, or 10.4 per cent, while in a group of 200 normal persons 71, or 35.5 per cent, reported cerebral injuries. The authors therefore feel that the argument for trauma must be abandoned in this group of tumors.

Tumors of the eye form a special group which, because of their infrequency, should be studied thoroughly and careful reports on the part of eye surgeons may add greatly to our knowledge of the importance of trauma. This fact has been pointed out by Stieren,[18] who has collected from the literature a group of cases in which trauma was mentioned by the patients. The interval between the injury and the discovery of a tumor in these cases was eight months, three months, sixteen years, one week, three years, three years, and in Stieren's case, nine years. The injuries consisted in blows and lacerations of some portion of the eyeball, probable in 2 cases. The discussion which followed was somewhat critical as to any correlation for it was felt by some of those present that the injury may have only set up a chronic irritation which gave opportunity for the later development of a neoplasm.

Recently Bonnet and Paufique[19] have reported a case of melanosarcoma developing in an eye which had sustained a severe blow sufficient to cause separation of the retina two years before.

Carcinoma of the internal organs, especially the stomach and pancreas, with trauma as the etiological agent, has again been mentioned in the literature only infrequently. A traumatic gastric cancer is reported by Luckow:[20]

> In a man wounded by shrapnel in 1917, examined because of epigastric pain in 1928, the *x*-ray showed splinters of metal in the liver, gall bladder and the greater curvature of the stomach. Laparotomy showed an extensive gastric cancer which had developed not around the metal fragments on the greater curvature, but where such tumors most frequently develop, in the pyloric region. This would seem to indicate that the metal fragments had nothing to do with the carcinoma. If the splinters caused the gastric cancer, why should they not have caused cancer of the liver and gall bladder?

The question of the possible production of tumors of the lymphogranuloma group has been raised. For example, S. Hirsch[21] in a detailed legal opinion stated that in a case of Hodgkin's disease the process was initiated or if not, definitely hastened by an injury. However, lymphogranulomatosis is somewhat foreign to the discussion as only a few pathologists regard it as a tumor.

The study of the relationship between trauma and leucemia will probably attract more attention in the future than in the past as the newspaper publicity recently given the disease has stimulated lay interest. Acute leucemia, though probably infectious in origin, will now presumably be associated in its inception with some accident; especially since children sustain an enormous number of both major and minor injuries this factor can always be invoked as a cause. Norikoff[22] has reported a patient receiving a fracture of a rib in

the region of the spleen which was followed one year later by chronic lymphatic leucemia. P. Keller[23] has also described a case of myeloid leucemia with a history of injury. All this seems rather vague as the spleen is not the site of origin of the disease. Not very convincing also is the case related by Olshausen[24] where a man developed a myelosis two years after a fracture of a lumbar vertebra. The example of plasma-cell myeloma occurring in the upper jaw of the patient of Rocquefer and Ruck[25] and cited as traumatic was on the cheek opposite to the traumatized area, and therefore can scarcely be included among the cases in which such an etiology is even remotely possible.

Verdelet[26] has also reported a lymphosarcoma of the axilla occurring in a man twenty-two years of age, six months after trauma to the chest and shoulder, but without any concrete evidence that the two were related.

The case of so-called traumatic lymphatic leucemia described by Jármai[27] occurring in a dog following injury is inadmissible on the basis of the blood counts as reported, since the leucocytes did not exceed 21,600 with 62 per cent polynuclears, 30 per cent lymphocytes, and 7 per cent monocytes. These counts continued for eight weeks at approximately the same level and the liver, spleen, intestinal follicles and nodes were enlarged. In man the diagnosis of leucemia would be improbable from these data. In fact the author states that the trauma could not have been the final cause of the leucemia but that if cellular proliferation was already under way the injury might stimulate the cell growth and thus make the leucemic process evident.

Ardoin[28] in an interesting report of a chordoma with numerous metastases has assumed without any definite evidence that since his patient was traumatized this was the exciting cause of the growth of the tumor. He cites four similar examples from the literature. Stanton[29] also reports two examples of chordoma with a history of injury two and four years previously but does not greatly stress the fact etiologically. Chordomata are so rare that a few observations can have no statistical value even in suggesting that the trauma stimulated a pre-existent tissue. No one would object to the possibility of trauma causing metastasis at a distance after the experimental work of Tyzzer[30] and Knox,[31] and the disastrous results observed after the attempts by charlatans to dissipate tumors by violent manipulation methods.

In connection with the melanomas there always arises the question as to whether a single injury applied to a preexisting benign tumor, such as a nevus, bears a definite relationship to a later development of malignancy. Fenwick and Wells[32] described the case of a girl having a small non-pigmented nevus in the temporofrontal region injured by a blow some six weeks before seeking advice. Nine months later she died with generalized metastases despite radical surgical removal and heavy radiation. The authors assume that the mechanism of the development of this malignancy is by way of injury to the trophic nerve supply. The weak point in the argument is that the nevus was evidently malignant before the injury and the latter only stimulated the growth and probably distributed the neoplastic cells inciting a malignant change in a quiescent growth. Fabre and Gadrat[33] described a similar case of a man twenty-eight years old who sustained an injury to a nevus on the chest and several months later a large fibrosarcoma had developed.

It is in the group of osteogenic sarcomas and giant-cell tumors that the question of trauma assumes perhaps its greatest importance, since the etiology of this group occurring in the extremities, and therefore subject to injury, provides a constant challenge to our knowledge of tumors. The excellent paper by Troell[34] includes a careful analysis of 78 cases which have been described as sarcoma

of bone. Troell is not at all deceived by the *post hoc, propter hoc* argument but has tried to analyze the cases in a logical manner. He concludes that only 59 of these cases are truly sarcoma, and in 24 of them there is a history of injury to the bone in the region of the growth, but even these 24 contained so many doubtful statements that only 9 would bear ultimate analysis. It must be remembered, as is explained by Troell, that many cases at first diagnosed sarcoma even from histological and radiological evidence do not prove to be malignant tumors, but very frequently are essentially benign lesions, reparative processes or other diseases. This greatly confuses the issue in records that have been made by enthusiastic observers even before the patient has been observed long enough for the actual progress of the disease to be understood. A case of bone sarcoma is reported by Cahen:[35] A man of seventy-nine sustained a severe blow on the right tibial tuberosity, very rapid growth taking place so that in three weeks the tumor was ten times the circumference of the normal bone. Eventually amputation was necessary, the tumor obviously becoming a malignant one.

Haler[36] has reported a synovial sarcoma of the knee:

> A woman of forty-one "jumped off a chair" and felt severe pain in the knee. The joint was explored seven months after the injury and found to be normal except for the medial meniscus which was ruptured. This was excised and the joint closed. Four months later the leg was amputated. The joint was found to be filled with a waxy homogeneous growth which invaded the patella. Microscopic diagnosis was round- and spindle-cell sarcoma, probably synovial in origin. In this case the exploration might logically be blamed rather than the first injury; in other words, neither was responsible for the tumor.
>
> The second case was that of a man of thirty-seven who struck his leg. The swelling never went down and two months later a biopsy revealed a spindle-cell sarcoma. The author is careful to say that the growths were associated with trauma, not caused by it.

The cases reported by Byron H. Jackson[37] were undoubtedly carefully studied and the author suggests that they may have some relation to trauma.

> One was a miner who sustained a contusion on both femurs by falling across a rail. Two months afterwards pain was still present in the left femur and the radiograph showed a periosteal osteogenic sarcoma, which however had no microscopic confirmation as the patient refused operation.
>
> The second case was a child of thirteen who three weeks before examination struck his knee against a hydrant. A tibial osteogenic sarcoma, microscopically verified, was found.

These and a number of other cases less well observed still remain baffling but important observations, and only by the collection of a large amount of such material can the importance of trauma be positively ascertained. Statistically there is no reason to believe that trauma of this type is more than an incident, since the number of traumatic lesions sustained by any individual, even those causing necrosis, hemorrhage, thrombosis and regeneration, is too numerous even for compilation. A patient's statement that he believes a tumor to be due to trauma, while to be recorded with respect, cannot be considered a contribution to the subject. Anyone accustomed to history-taking is well aware of the fact that a patient nearly always has an explanation for every symptom and disease, and an affect is often present regarding these causes which the doctor does well to leave undisturbed. There can be no question that trauma in the form of sudden shock or over-exertion is an extremely important agent in the production of a decompensated heart, the localization of certain types of abscesses and tuberculous foci, but the explanation of the patient regarding these matters, while intended to be honest, is often wrong. No doctor assumes that a patient knows that he has vegetations on his heart valve, and the embolic result of these vegetations is entirely outside the patient's ability to predict or understand. No

matter how good his intentions, his observations and explanations are at fault. A patient cannot be expected to know whether the pain which he feels in his upper abdomen has originated from above or below the diaphragm, but the doctor regards it as his obligation to ascertain this fact, regardless of the patient's opinion. Therefore it seems remarkable that many physicians attach such great importance to the patient's interpretation of the cause and time of origin of his tumor.

## CONCLUSION

In conclusion, it may be said that the general trend of medical thought has probably been influenced in the last five years, perhaps unconsciously, by the legal and compensation verdicts which have been returned. On the other hand, medicine has contributed a considerable number of useful observations, but has offered no definitive answer to the question of the relationship of trauma and tumor. Probably the most valuable work which can be done in this field is to educate the medical profession toward more complete factual case histories in which personal opinions of an inexpert type either as regards the interpretation of clinical observations, radiographs, or pathological slides should take second place, and in each of these fields consultations from a number of experienced persons should be applied to an understanding of the individual problem. From this aspect the practice of some of the German writers who record in great detail the very complete physical examination made of the injured person together with the names and opinions of the experts who have made the pathological diagnosis on the microscopic slides of the tumor under discussion is a good example too infrequently followed.

Finally, the writer believes that no valid evidence has been offered since 1929 to change the opinion expressed in a more extensive survey of this subject published at that time that as yet no causal relationship has been established between a single trauma to healthy tissues and the appearance of a malignant neoplasm.

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# TRAUMATIC PEPTIC ULCER*

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THE factor of trauma in the genesis of acute and chronic peptic ulcer has been of interest for many years. In spite of extensive German and French writings, which embody numerous reports of cases, contributions to the subject in English are strikingly few. However, some of the earlier Continental reports were written before roentgenoscopy came into use, and before duodenal ulcer was a frequently observed entity, and many times the diagnosis of ulcer was based on symptoms alone, without confirmation by operation or necropsy. But contemporary authors of texts on diseases of the stomach writing in English, such as Crohn, Hurst and Stewart, Rehfuss and others, have given consideration to the clinical significance of traumatic peptic ulcer. A complete review and appraisal of all material printed up to five or six years ago has been made by von Redwitz and Fuss. The 1930 edition of Stern's textbook also contains a comprehensive review of the German literature.

Von Redwitz and Fuss concluded that undoubtedly in rare instances, trauma may lead to the origin and development of a chronic ulcer of the upper part of the digestive tract. By virtue of the excellent tendency to heal demonstrated after a freshly sustained injury to the gastric mucosa, only under circumstances entirely favorable to formation of ulcer can acute trauma result in a chronic ulcer. Therefore, it must be assumed that other ulcer-forming factors are present; acute trauma really is probably more a precipitating than an unaided causative factor. It would be difficult to determine whether death of a circumscribed area of mucous membrane had resulted from direct injury to the mucous membrane itself, or whether the necrosis had been caused indirectly, by injury to blood vessels and nerves. It appears more probable that chronic trauma has a definite influence on the chronicity of an ulcer, but it does not always suffice of itself to produce the picture of chronic peptic ulcer.

From a clinical standpoint it is obvious that trauma is not a factor in the origin of the majority of acute and chronic gastric and duodenal ulcers, unless the incidence and significance of mild degrees of trauma to patients predisposed to ulcer has been disregarded. Because there is no indubitable proof that an ulcer forms as the direct result of trauma, there are physicians who, without due deliberation of all the ascertainable facts, deny the existence of a traumatic ulcer under any circumstance. Trauma, when it is said to be causative of ulcer, usually implies external blunt force directed to the epigastrium, without direct penetration of the abdominal or visceral parietes, resulting in variable degrees of injury to the gastric wall. It is chiefly in this sense that the subject will be considered; a case also will be reported. However, mechanical trauma to the stomach and duodenum may occur not only from without, in an acute or chronic fashion, but also from within in the same way. Aside from ulcers resulting from external trauma, the other types of traumatic ulcer chiefly observed clinically are those secondary to foreign bodies, and diaphragmatic hernia.

## ULCER THE RESULT OF EXTERNAL VIOLENCE

Experiments on animals and repeated observations on man have shown con-

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clusively that a severe blow to the upper part of the abdomen may result in rupture of the viscus, especially if the stomach is full, or in single or multiple defects of the gastric mucous membrane, consequent on laceration. This paper is not concerned with rupture of the viscera, and the question to be considered here is whether laceration of the mucous membrane can be regarded as the preliminary stage of ulcer.

To Potain is generally given the credit of first describing traumatic ulcer, in 1856. The external violence may be sufficiently great to produce multiple acute gastric ulcers, as well as rupture of other viscera, especially the spleen, or liver, or both, as in a case reported recently by Hausbrandt. On the other hand, the injury may be much less severe, and may be followed promptly by the onset of symptoms and signs of peptic ulcer; these symptoms and signs may not have existed before the injury, and sooner or later they may be attributed to a demonstrable chronic lesion in the stomach or duodenum, especially in the former. It is this latter type of case that is of greatest interest from a scientific and medicolegal standpoint, as will appear.

The conclusion that injury was the cause of a given chronic lesion must be drawn with considerable circumspection, for it is generally known that purposeful injuries to gastric mucous membrane in the experimental animal are followed by rapid healing. Moreover, the gastric mucosa of man can stand much surgical manipulation, and yet smooth healing invariably results. Corrosion with chemicals, accidental or intentional, lead chiefly to formation of cicatrices and stenosis, without actual ulcer. With rare exceptions, repeated injury to the stomach by instrumentation has no ulcerative sequelae. Then there are numerous reports of cases, dealing with necropsy or surgical procedures on professional sword-swallowers, or on hysterical or insane people whose stomachs contained knife blades, forks, needles, hairpins and other sharply pointed objects. These objects often had remained in the stomach for a long time without producing the characteristic round ulcer. The latency of many gastric and duodenal ulcers is a proved fact, and objectors to the hypothesis that trauma causes ulcer rightly point out that trauma may activate a preëxistent asymptomatic lesion, just as it may prematurely provoke hemorrhage from, or perforation of, a chronic lesion. For example, Rudge's experience is not in accord with the viewpoint of those who accept the traumatic origin of peptic ulcer, although in a few cases he has observed that trauma may play a part in certain preëxisting or predisposing complications such as perforation and hemorrhage. All these valid objections, coupled with the fact that there is rarely a history of significant trauma in the usual case of peptic ulcer, argues for skepticism regarding the direct relationship of trauma to the lesion. Many writers look on trauma as the predisposing cause, and believe that other pathogenic factors, such as hereditary influences or constitutional predisposition, injury to nerves or vessels, hyperchlorhydria, infection, nutritional disturbances, and so forth play the chief part in production of the chronic lesion. Undoubtedly one of these factors is operative or perhaps more than one, but which one assumes major importance is difficult to say, in the light of present knowledge.

In this connection the recent work of Smith and McConkey is of interest. Mechanical injury to the mucosa of the duodenum of a guinea pig that was fed on an adequate diet was followed by rapid and complete healing, whereas a similar injury to guinea pigs that were fed on a diet deficient in vitamin C resulted in the formation of peptic ulcers. Diets deficient in vitamins A, B, and D did not cause peptic ulcers, if the supply of vitamin C was adequate. To just what extent these observations apply to traumatic ulcer of man is questionable, for the factor of vitamin deficiency, under normal economic conditions apparently plays a negligible, if any, part in the condition of our patients.

There are many authentic reports of cases that, notwithstanding all the foregoing, are convincing that trauma, under exceptional and rare circumstances, is basically, if not entirely, the cause of a chronic lesion, and certainly of acute and subacute lesions. All of the cases reported seem to have certain common clinical characteristics. Injury to the epigastric region is rather promptly followed by pain and hemorrhage, and gradually the symptoms partake more of those of ulcer. Eventually gross ulcer develops, and its presence is confirmed by operation, necropsy, or roentgenoscopic examination. One gets the impression that the injury produces acute ulceration, which is evidenced by immediate hemorrhage in some cases. If hemorrhage is delayed, it is reasonable to assume that the trauma produced a submucosal hemorrhage, resulting in a hematoma of variable depth and size, followed by necrosis, proteolysis and eventually crateriform ulcer.

Potain's report concerned a woman sixty years of age, on whose abdomen a heavy piece of furniture fell. Three days thereafter, pain, hematemesis and vomiting ensued. A variable course of eight years ended in her death. Necropsy revealed an hour-glass constriction of the stomach as the result of an old ulcerative, cicatrized lesion.

Kemp's patient was a healthy woman, aged twenty-two years, who was thrown from a trolley car, and her abdomen was struck in the epigastric region. Pain and tenderness were present at this point and persisted for six weeks, when severe hematemesis suddenly occurred. This was eventually followed by symptoms of peptic ulcer, which were permanently relieved by treatment within eight months after the injury was sustained.

Hurst and Stewart reported the case of a man who was injured by coal falling on his epigastrium. A diagnosis of ulcer was corroborated by roentgenographic studies. Within three weeks the ulcer had undergone perforation.

Ramond and Chêne also reported two convincing cases. One case was that of a man, aged thirty-eight years, who at operation fifteen months after injury was found to have a chronic ulcer, with much scarring, hour-glass constriction, and extensive perigastritis. The other case was that of a woman, aged thirty-three years, who twelve years previously, had been hooked in the epigastric region by a cow. Within half an hour after the injury she vomited blood. There was moderate hematemesis for three weeks thereafter. Epigastric pain, with projection of the pain to the back, epigastric burning, and loss of weight were also noted. Roentgenoscopic examination revealed a niche on the lesser curvature, with considerable retention of barium after six hours. At operation the ulcer was found to be crateriform and to have penetrated the pancreas. In spite of extensive adhesions and a difficult operation the patient made a good recovery.

Gerendasy reported the case of a widow, aged forty-four years, who sustained a severe blow to the epigastric region while riding on a motor bus, but who was able to proceed home. The following day she experienced vague epigastric distress, and on evacuation of the bowels she observed that the stools were black. In less than twenty-four hours afterward, she had nausea and hematemesis. During the patient's stay of several weeks in hospital there were repeated hematemesis and melena. Soon after the accident, examination revealed contusion and bruising of the abdominal wall just below the ninth right costal cartilage. Three days later the value for hemoglobin was 68 per cent, and erythrocytes numbered 3,250,000 in each cubic millimeter of blood. Two weeks after the accident roentgenoscopic examination gave evidence of a small, well-defined niche on the lesser curvature of the first portion of the duodenum. After a total of three weeks' care in the hospital the patient remained free of symptoms for five weeks, following which time symptoms of ulcer recurred periodically, along with an episode of mild hematemesis and melena. Marked epigastric tenderness, slightly to the right of the median line, persisted. Subsequent roentgenologic examination revealed a deformity characteristic of a penetrating duodenal ulcer. At no time prior to the accident had she had any symptoms referable to the upper part of the digestive tract.

We herewith submit a report of the most recent case of gastric ulcer following trauma that has come under our observation:

A farmer boy, aged sixteen years, was brought to the clinic in an ambulance, March 18, 1932. Except for mild attacks of influenza and tonsillitis he had enjoyed excellent health until October, 1931, when he had fallen headlong from the back of a moving automobile, striking the epigastrium forcibly on an empty spare tire rack. He was knocked to the ground and was breathless for thirty seconds or longer. At that time he had experienced no actual pain although there was some contusion of the upper left abdominal wall and he did not feel well for the remainder of the day. Within twenty-four hours epigastric pain appeared and was most marked on exertion, so that he eventually was compelled to discontinue all work, attendance at school and participation in sports. Aggravation of the pain was not caused by taking food. Shortly after the injury he had had occasional left epigastric pain which appeared half an hour to an hour after meals, lasted for an hour or so, and disappeared before the next meal, so that he was always free of pain at meal time. Five months had passed since the injury, the foregoing symptoms persisting off and on, when, after a heavy day's work on the farm, he had recurrence of epigastric pain and noticed tarry stools. On the two following days he was free of pain. Then the pain became more marked and was associated with thirst, massive hemorrhage from the stomach, syncope, and shock. Following this hemorrhage the pain disappeared, and the boy was brought to the clinic.

Fig. 1. Niche of penetrating gastric ulcer on lesser curvature, above incisura angularis.

The patient was pale, in a fair state of nutrition, with no abnormality other than anemia (hemoglobin, 68 per cent, and erythrocytes 3,860,000 in each cubic millimeter of blood) and moderate tenderness, without rigidity, in the left side of the epigastrium. Roentgenoscopic examination, March 21, 1932, gave clear evidence of a penetrating ulcer on the

lesser curvature, above the incisura angularis. Three days later, after administration of histamine, there was a great deal of secretion, able so far to get him to return for reexamination, although he lives just across the border of a neighboring state.

FIG. 2. Same case. No demonstrable gastric defect after three weeks of intensive treatment in hospital.

maximal total acidity was 106 units, and free hydrochloric acid, 94 units (titration with tenth normal sodium hydroxide). Because the patient was young and the symptoms of recent onset, operation was deferred and intensive treatment undertaken. He was placed in hospital and made a favorable response. Successive roentgenographic examinations disclosed gradual diminution in the niche, and April 16, 1932, no defect was demonstrable (Figs. 1 and 2). The boy was dismissed to follow a regimen for ambulant patients with an ulcer. He has been so well since that time that we have not been

The fact that the usual type of chronic gastric ulcer is uncommon in youths, and that the ulcer promptly disappeared under treatment, tends to support our opinions that the lesion was of recent and traumatic origin.

### ULCER THE RESULT OF INTERNAL TRAUMA

In the past, numerous reports have been made of ulcer appearing secondary to foreign bodies in the stomach. Some of

these reports cannot be accepted without reservation, for the diagnosis of ulcer was based entirely on symptoms, which may be

diaphragmatic hernia. Harrington, who has had large experience with such hernias, has stated that erosions and occasional

Fig. 3. Niche caused by large chronic ulcer in posterior wall of stomach, near lesser curvature, and filling defect in antrum, produced by large hair ball.

fallacious. This subject has been well covered in the articles of Wölfler and Lieblein and of Rivers and Davison. Surgeons of wide experience have reported instances of ulceration, apparently the result of retained unabsorbable suture material or of the presence of a Murphy button. We have in our collection an instance of ulcer provoked by a hair-ball. The patient was operated on by Balfour and the case was reported by Waller (Fig. 3). Schulze reported 5 instances of ulcer secondary to persimmon bezoar and added one case of his own. Of more clinical importance is the association of ulcer with

ulcers are more likely to occur in association with the congenital form of diaphragmatic hernia in which the herniation takes place through the esophageal hiatus, rather than in the traumatic form. In the latter the stomach usually becomes transfixed by adhesions, which prevent the sliding, chafing and semirotatory movements possible with congenital hernias. Erosion of the mucous membrane occurs at the point of angulation and fixation to the hernial opening in the diaphragm or to structures in the thorax. In chronic cases this erosion may progress to definite ulceration and may present some of the

symptoms of gastric ulcer. Roentgenograms illustrating such a case have been published by Harrington. Truesdale has recently collected from the literature a series of 17 reports of gastric ulcer associated with diaphragmatic hernia. Fifteen of these hernias were of the congenital type. Some authors regard the ulcer as merely a coincidental lesion, but we are inclined to consider it as of traumatic origin in most instances, because it disappears promptly and permanently after successful reduction of the hernia, without any special treatment directed against the lesion. Although there may be no anemia in the presence of single or multiple ulcers, we feel that most cases of secondary anemia in association with diaphragmatic hernia are directly the result of occult or gross bleeding from erosion or ulceration. This opinion is not altered by the fact that the ulcers may not be discoverable by roentgenoscopic examination and may not give rise to symptoms.

Recently a patient presented himself chiefly because of marked secondary anemia. The persistent presence of occult blood in the feces led to the discovery of a congenital diaphragmatic hernia and ulceration of the gastric mucous membrane.

## MEDICOLEGAL CONSIDERATIONS

Persons without medical experience are prone to attribute to injury or accident the genesis of gastrointestinal disturbances. The fact that such disturbances may have antedated the injury may be minimized or the information may be withheld. All who specialize in gastroenterology receive numerous requests to express an opinion in general, or in individual cases, concerning the relation of external trauma to gastric disorder. In these days of increasing numbers of automobile accidents, the medicolegal and scientific aspects of the subject take on added importance. In the event of litigation it might be useful to keep in mind the four postulates which Lininger and Molineus have declared claimants for compensation should satisfy before their claims can be considered valid. The postulates are as follows: (1) Proof must be given of the absence of gastric disease prior to the accident. (2) The injury must have been sufficiently severe to cause the lesion, and the trauma must be localized to the epigastrium. (3) The symptoms must have followed the injury immediately. (4) There must be continuation of symptoms and signs that point to gastric or duodenal ulcer.

Some of the German authors, on the basis of numerous reports, have expressed the belief that the traumatic nature of the lesion may sometimes be established at necropsy or at operation. The nature of the pathologic findings naturally varies with the extent of the trauma and the duration of the injury. Published reports have disclosed that formation of scars and pyloric stenosis, without ulceration, are frequently noted in advanced cases. Hausbrandt expressed the belief that if a lesion can be examined either in the living patient, or after his death, within one to two weeks after infliction of the injury, one will find it fundamentally different from a peptic ulcer. In his opinion, the lesion is sufficiently characterized by its irregular, notched margins, the undermining of its entire border, and frequently by its atypical situation. Fresh lacerations are recognized immediately. Even soon after injury, if a typical round ulcer is found, it is certain that the lesion is not traumatic. Later on, however, after the traumatic lesion has developed into a chronic one, distinction is frequently impossible on the basis of the features described. Hausbrandt contended that only if the ulcer is found in a long scar which evidently resulted from laceration of the mucous membrane, can it be concluded with certainty that it was of traumatic origin.

## SUMMARY AND CONCLUSIONS

There is apparently adequate evidence to justify the contention that under exceptional circumstances a chronic ulcer

of the stomach can have its origin in external, non-penetrating trauma to the epigastric region. In one case, fairly characteristic symptoms of a hemorrhagic gastric ulcer developed following a severe blow to the left epigastric region. Roentgenoscopy confirmed the presence of a penetrating ulcer near the lesser curvative five months after the injury. Following hospitalization and intensive medical treatment, the lesion completely disappeared and clinical cure resulted. In any case in which a plaintiff claims that a gastric or duodenal ulcer followed external trauma, the physician whose opinion is sought should see that the four postulates of Liniger and Molineus are satisfied.

A second cause of ulcer is chronic trauma from within. The commonest form of ulcer of this nature is that secondary to congenital (non-traumatic) diaphragmatic hernia; foreign bodies in the stomach may also cause ulcer. Such secondary lesions are not indurated as a rule and all tend to heal readily following reduction of the hernia or removal of the foreign body.

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# TRAUMA AND THE AGED PATIENT*

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IT is axiomatic that patients of advanced age are poorer surgical risks than those of less advanced age. This is true in general surgery and especially true in traumatic surgery. However there are certain factors irrespective of the calendar rating which are of major importance. We have all had the experience of treating patients of sixty-five plus who were better subjects than others of forty-five plus. Hence we are confronted by the query: What constitutes age from a physical standpoint? We can attempt an answer by narrating some of the elements predetermining *premature* aging. Here is a division that is of primary import, for the one is *physiologic* and the other *pathologic*. I would postulate this problem by narrating the following as basic considerations:

1. *Heredity*. There is no question as to the viability of one strain by comparison with another strain. Ancestry is an element of primary importance, and longevity in forbears is admittedly a potent determining factor.

2. *Prior Diseases*. It is conceded that any illness capable of producing appreciable symptoms such as fever, cardiorespiratory, vascular-renal, gastrointestinal, or neural involvement, inevitably registers by pathological markings. This being interpreted means that the viscera affected are more or less permanently damaged; they are, so to speak, traumatized; they are no longer a perfect mechanism. To be sure, the reparative power may be almost absolute, complete; but nevertheless, there has been an attack with a resultant effect, microscopic or macroscopic. It is beyond dispute that certain of the so-called diseases of childhood initiate the process leading to premature aging. Scarlet fever, measles, diphtheria are examples of the less powerful; rheumatism or pneumonia examples of the more powerful. In later life, influenza, typhoid, gastrointestinal and urogenital diseases all add their quota. The venereal group, syphilis especially, are important precursors. It has been said that tuberculosis registers in the vast majority of human kind because autopsies with great regularity disclose demonstrable evidences in the form of scars or adhesions.

3. *Habits*. Overstrain or overtaxing of the human machine is reasonably certain to lead to pathological changes in the majority, despite compensatory benefits conferred upon the minority. The effects of overactivity in sports, of too strenuous work, of malnutrition are important factors, especially if linked with over-indulgence in alcohol, tobacco, tea, coffee or venery. Long ago Osler referred to those who too diligently worshipped at the shrine of Bacchus, Venus and Vulcan as presaging an unwilling transfer of affection in some cases to Mercury.

If then heredity, prior disease and habits are anticipatory factors, we can go further in our attempt to delineate the pattern that we eventually recognize as age markings. What then is age? From a *physiologic* standpoint it is the quantitative measure of time displayed in *retarded* function. From a *pathologic* standpoint it is the qualitative measure of time displayed in *damaged* function. The one is the natural manifestation of wear and tear, the aging process toward senescence; the other is the unnatural manifestation of actual damage progressing toward senility. One is physiologic, the other pathologic. One is heritage; the other acquisition; one is inevitable, the other inescapable.

If then this is the prelude, what are some of the elements making of the subject what

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we call a poor surgical risk? It is conceded that the cardio vascular-renal mechanism is for us the most vital, and alterations in this circuit constitute a major hazard; in effect so changes the age structure of the patient that the calendar is no longer an actual guide as to the real age of the patient. This is equivalent to stating that our patient is in reality as old as his tissues, even though in decades the contrary is asserted. It is an old saying that a person is as old as the arteries; but we as traumatic surgeons are inclined to believe the patient is as old as the neck of the femur.

Given then a patient aged from a physiologic or pathologic standpoint, let us if possible rate potential or actual defects in relative importance. If you had to choose for an aged patient the least important systemic defect, what would you select? And if you had to choose the most important, what is your selection? Here is a contrast in selectivity, a ratio as between evils, a computation as to hazards.

For my ideal aged patient, give me first that perennial youthful state of mind which radiates faith, hope, confidence, and which is linked by years of tested experience to cooperate with order and system and discipline. Give me these for my aged patient the victim of trauma, for thereafter the physical handicaps of maturity or co-existing pathologic processes are vastly less important.

In trauma, the cooperation of the patient is more needful than in any other type of surgery. You enjoin your laparotomy patient to remain quiet; you beseech your traumatic patient to become active. Immobility in the one; mobility in the other. Next give me for the ideal, a cardiovascular-renal system not too compromised. Next in this idealistic array, give me adequate respiratory capacity. Hence my acme for an aged patient is a youthful state of mind and a relatively unblemished cardiovascular-renal-respiratory system. Purposely I have not stressed the gastrointestinal-portal system because it is to me the least important, and if damaged is perhaps the easiest to control. If these are the desirables, then the reverse are the undesirables, and we are all willing to admit the handicaps imposed by a senile state of mind and a damaged cardiovascular-renal-respiratory system. Add to this hazard, diabetes and arthritis and then surely the outlook is poor. Perhaps it may be well to portray the ideal physique as a part of the problem. Give me the wiry type of person who has never been obese; the actively alert individual accustomed to the out-of-doors; the do-it-myself type who has not been pampered by service and who has arrived as the result of self endeavor and a resolution to get on despite hardships. This is what I call the "wren" type, a chirruping, flitting, active, foraging brood, always on the go and going all ways. This is the grandmother or grandfather type familiar to us all, the envy and despair of the flapper brand of their children who have been brought up in this mechanical push-the-button era and who in all their lives perhaps never had to climb more than one flight of stairs in any given journey, and who started with the perambulator and continued with the automobile or aeroplane.

This then is the structural background for our traumapathies, and now let us survey some of the essential types of injury and their effects on the aged.

1. *Shock.* The importance of this is in terms of likely severity, and equally important because it delays definitive treatment beyond what I have chosen to call the "golden hours" for traumatotherapy, namely, the first six hours posttrauma. We know that wounds treated in that interval are unlikely to show severe infection, and that fractures are then the more easily reduced. If we are forced to wait beyond this six hour period, complications arise and new factors are introduced.

2. *Wounds.* The reparative power of aged integument is of course less than that of more youthful integument. Again, sclerosis of vessels adds that possibility of phlebitis, thrombosis and embolism. Ulcer-

ation and gangrene are more readily induced, and if infection ensues, the probability of local or distant spreading is an imminent hazard. Recumbency is often in and of itself a source of impaired circulation leading to hypostasis, especially if infection coexists. Further, if anesthesia is required, an additional burden is imposed and thus from every standpoint a relatively simple laceration may assume grave proportions.

3. *Fractures.* Healing power is again delayed or diminished, although I recall a patient of ninety-two who walked after sustaining a fracture of the neck of the femur and she died in her 100th year. The oldest patient I ever saw was 102 with an inter-trochanteric fracture of the femur and in consultation with her physician the main question was whether or not it was safe to allow her out of bed. I said it was, and next day she got up and stayed out of bed nine hours, thus proving that it is hard to keep a good woman down!

The management of the aged with a broken bone introduces many factors quite unrelated to the traumapathy itself, for we are concerned not alone with the initiating element but also with what may be called the entire body politic. Here again we may find it impossible to impose definitive treatment because shock prevents; and to defer reduction we know may infer subsequent impairment or actual defeat. Here we must determine on a line of procedure quite different from what we might prefer, because we realize that our problem is complex and that the treatment of the involved person is more important than the treatment of the involved part. Our aim in all traumata is to prevent what I call the three D's, namely death, deformity, disability. In the aged this precept is all compelling and our experience teaches us to be wary. Hence we temporize to test out the reaction of the patient in recumbency, and for that reason we do not resort to heroics applicable to a better risk. In this class of patient we know that the complete immobilization of a limb may be excellent for that broken bone but fatal for that broken patient. Hence we do not rush the hill in high but drop back into second speed until we reach a level spot, and perhaps coast a little in neutral when chance provides the opportunity. Now this applies to a closed fracture of one bone, but with greater precaution applies to an open fracture, or to multiple fractures. The question of anesthesia enters also, and in the lower extremity we will probably prefer spinal to inhalation anesthesia, even if gas oxygen alone suffices.

With systemic anesthesia of the morphine or avertin type, we may have useful allies; but again caution is necessary. The point is that we should keep in a state of awareness, realizing that to do otherwise is to invite a dramatic catastrophe.

Within recent weeks I had a sixty-eight year old patient who appeared ten years younger than her calendar age. I did an easy open correction of a comminuted closed fracture of the tibia and fibula, applying a circular plaster casing. She reacted well from her short anesthesia, but within an hour after she left the operating room she suddenly became dyspneic, cyanosed, and died. An experience like that, unusual as it is, gives pause to like endeavor under similar conditions.

Fracture of the neck of the femur is the typical bony injury of the aged. We in our Reconstruction Hospital service however well known that this lesion is by no means confined to old patients, and our recent *Traumatology* leaflet records a case in a six year old girl. The fractured hip problem of the aged person is one that challenges the managerial skill of all of us. We are urged by some to immobilize all of these patients in a plaster spica, the statement being that shock is thereby lessened, nursing care is aided and eventual cure is more likely than by any other procedure. Recently the operative cure for this fracture has had many exponents, others advise bone grafting, others bone drilling. Again others assert the value of traction with or without suspension. And finally there are

those who suggest an ambulatory splint, or even a do-nothing-at-all procedure because a poor end-result by any method is the rule and not the exception. Here then is a wide choice, and it may be expedient to grade our aged patient in an attempt to appraise the best type of treatment for this or any other trauma to which our aged patient is subjected. Again let me insist that "aged" to me has no calendar significance and that age in years is less important than age in tissues. In other words, "aged" is in terms of tissue reaction and not calendar reaction; a patient is as old as his tissues and not as old as his years. For appraisal purposes the following is suggested, based on the three constitutional criteria previously stressed.

In this arbitrary classification, this assay, I would rate *Heredity* as having a potential sales value of 30 per cent, *Prior Diseases* at 50 per cent, and *Habits* at 20 per cent; that is, if all of these three factors were top notch, the sum of them would give our patient a 100 per cent chance. Hence in the A group of trauma risk I would place the most favorable, in the B group those in the mid group; and in the C group the ultra-hazards. A is the good risk group; B the fair risk; C the poor risk.

Let it be stated that *Heredity* encompasses race, sex, station in life and other factors that may be important.

*Prior diseases* especially stresses the cardio-vascular-renal group already mentioned.

*Habits* includes type and amount of work or play, as well as the prior drinking, smoking, venereal practices of our patient, and also the all-important state of mind.

Now of course there is no sense in trying to make this or any other classification hard and fast, for that cannot be done even by a surgical pawnbroker who is acting as appraiser; but at least it is a sort of physical income tax that may aid in computing the hazards ahead.

If our patient has an excellent *Heredity* we rate 30 per cent for that item; if *Prior Diseases* have not left indelible pathologic imprints, then we rate that item 50 per cent; and if the *Habits* rate to the maximum we allot 20 per cent, and thus the sum would be 100 per cent.

Those in the 75–100 per cent group I would rate as Class A.

Those from 50–75 per cent group I would rate as Class B.

Those from 25–50 per cent group I would rate as Class C.

Obviously most of our patients will be in the B and C groups, and the endeavor is to have some common denominator for appraisal in terms of figures rather than in terms of words. The income tax statisticians are not interested in any set of words that seek to portray wordly goods by any such terms as "rich," "moderate" or "poor" circumstances, and I submit that a physical appraisal in mathematical terms is more accurate than any delineation in language terms.

If thus appraised, we can the more accurately assign our patient into the appropriate bracket for treatment, having always in mind the initial severity of the injury. Of course it is self-evident that an aged patient may sustain just as irreparable injury as a younger patient; but the point sought to be stressed is that age in and of itself does not necessarily consign the patient to the permanently disabled grouping. Naturally our tendency is to consign or assign our aged patients into the derelict class, but we all have had the happy experience of realizing that age of itself is not a barrier to recovery if balanced by some of the factors just narrated.

There are some other factors of importance from a clinical standpoint, and I would call attention to the importance of *osteoartbritis* in joint injury, notably when the spine is affected as in back strain or some associates. It is probably true that most persons over fifty show by *x*-ray or clinically some signs of osteoarthritis, and this may be relatively benign until activated by injury or rendered more immobile by enforced inactivity. I am convinced that a number of our arthritics are not so

much damaged by reactivation of an existing arthritis as they are damaged by a diminution of the articular circulation due to remaining inactive. It is, so to speak, articular hypostasis, the analog of the hypostatic pulmonary stasis we so zealously seek to prevent.

*Head injury* in the aged may have a greater ulterior effect than in the younger group; but this not universally true, and the effect is in part at least often compensated by the lesser demands of an aged person, and by that habit of mind that so constantly asserts itself as to be almost automatic in that group, advanced in years to be sure, but also advanced in the will and zeal to follow on with the procession. Like veterans, another war is just another war, and not a new, unknown, terrifying exploit.

*Malignancy* from trauma is again front paged in some of the medical magazines. I am not in accord that injury is much of a factor in this direction, and certainly one injury, isolated, solitary, exceedingly rarely stands in causal relationship. That continued, repeated trauma may be a factor seems reasonably certain, but that also is very rare. If we take the incidence rate of cancer as an index, I am quite certain that the cases alleged to be due to trauma will be little if any higher. In my own experience with the injured, I can with difficulty recall only 2 cases in which trauma and injury appeared to be correlated. Let us not forget the law of averages, the doctrine of coincidence in this connection, and do not let us become stampeded by some instance that appears beyond question until we fully analyze. What usually and ordinarily happens under a given set of circumstances is our background, our foundation, our lexicon of experience that guards and guides in our opinion and judgment. Injury was once regarded as an etiologic factor in a great group of lesions, and the older text books rated alcohol, syphilis, gout and trauma as the quartette comprising the swan song as a prelude to decrepitude. But trauma was found to be out of tune so often that it was banished and now has a choral quality all its own. Perhaps this is a good place to say that traumatic surgery has achieved for itself at last a high place in the surgical realm, and no longer is our branch of surgery regarded as an alien nor are its devotees rated as menials in surgical craftsmanship. It may interest you to know that in 1917 when I tried to get the publishers to accept the title "Traumatic Surgery" for my book, they scoffed and said there was no such selective type of surgery. Today, practically every general medical magazine is narrating the experiences of men the world around in this branch of our calling.

And we have only begun to explore the field and just now, as in every development of our art, we are in the maze of apparatus and technical aides just as in years gone by there were special scalpels, special clamps, special gadgets of all sorts. But a good surgeon, like a good artist, a good musician, a good mechanic, a good sportsman, relies not so much upon the appliance in hand as upon the guidance imparted thereto by knowledge and experience.

And then there is the dispute concerning the relationship of trauma as an activating factor of coincident disease. Can diabetes be thus re-awakened? Can an ulcerative process of the leg in an arteriosclerotic be charged to a simple contusion of the shin? Can cerebral apoplexy follow a blow on the head? Can osteoarthritis of a joint or of the spine become aggravated by trauma? Can tumor of the brain be traumatic? Can cardiac decompensation occur from trauma? These are frequent allegations in medicolegal and compensation cases; but you know and I know that chronic disease has periods of accession and remission, and that in and of themselves they vary in their manifestations, quite apart from trauma of any other ascribed factor.

We none of us deny the probability of a relationship in special cases when the injury is adequate and the symptoms are adequate; but we know the relationship to be unusual, exceptional, rare, especially

when the sequence of symptoms as to site, type, time of appearance and progression is out of line with our previous clinical experience. Here again our opinion should be based on what usually and ordinarily happens, and we should not swerve from our background of accredited knowledge unless there is an actual and not a theoretical or argumentative basis therefor.

Trauma has criminal record enough without being charged with aiding and abetting crimes due to disease and other factors. So we must not let trauma become a scapegoat for too many pathological processes, even though the present evolution of our branch of surgery will continue for a time to attempt to fasten on trauma a responsibility that will later be properly allotted elsewhere.

Do you realize that tabes was once regarded as traumatic, likewise pulmonary tuberculosis and tubercular meningitis? And quite recently I read that gastric and duodenal ulcers were traumatic in origin. You do not believe so and neither do I; and we know that trauma occurs so often that if these things were traumatic there would be no need for argument because you and I would have the relationship indelibly impressed by an appropriate sequence of symptoms.

## SUMMARY

So to summarize, let me again stress the importance of regarding the aged patient not by the calendar of the years but by the calendar of the tissues. Let us assign our patient the A, B, or C rating based on a definite computation in respect to Heredity, Prior Disease and Habits. And let us not forget the lessons of our own experience and that of our predecessors in rendering to Caesar the things that are Caesar's and let us disassociate from trauma those things properly belonging elsewhere.

Finally, in the traumatotherapy of the aged, let us be more adaptable to the broken patient than to the broken part, remembering that a woman may be as old as she looks, but a man is never old until he quits looking.

# CHARACTERISTIC CONTACT BRUISES, ABRASIONS AND OTHER INJURIES IN AUTOMOBILE ACCIDENTS

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THE only excuse for the writing of this short excerpt under the heading of a paper, is the hope that the illustrations, per se, may warrant publication.

Figures, which are deplorable, show that some 30,000 deaths, and injuries to the extent of almost a million, result from automobile accidents each year. The costs entailed, though equally enormous, do not seem to have been directed toward the elimination of such accidents, save, in a small way, by the institution of safety patrols in schools, and, in an even smaller way, the propaganda against danger by a few isolated townships. Certainly the manufacturer, in his successful attempts to increase the speed of a car, adds little to the comfort of a belated pedestrian.

The obvious complications resulting from highway accidents require not only careful investigation in order that expert testimony in criminal, civil and compensation courts may be of value, but also forms, of necessity, a major part of the medical examiner's work.

The exigency of autopsying all victims of highway accidents, therefore, needs no further comment. The only exception, perhaps, is in children where traumatic injury is obvious; as, for example, the avulsion of brain tissue through a fracture of the skull; obviously the question of alcoholism is of no import.

In adults who are killed instantly or live but a few hours, the question of intoxication is pertinent. In our locality 20 to 25 per cent of pedestrians are inebriated when struck. As a routine, on all such cases in which the person is killed outright or dies within a few hours after the accident, 500 gm. of brain tissue are sent to the toxicologist for examination.

It is of interest that, though the external examination of the body reveals such marks as bruises, lacerations, abrasions, hematomas, fractures, etc., there is but seldom any special pattern from which we could positively affirm that the victim was struck or came into actual contact with any part of an automobile. The evidence in most cases, therefore, is only circumstantial and goes no further than the fact the body was exposed to external violence.

However, in the presence of fractured ribs, lacerations and blebs of acute interstitial emphysema over the free borders of the lungs and visceral pleura, together with other evidences of chest compression, it is reasonable to suppose that the vehicle had passed actually over the body; the fact that there may be no characteristic external marks, and in some cases none at all due to the protection afforded by heavy clothing, does not detract from such an assumption. Likewise, such a sequence of events is applicable to the abdomen where rupture of the spleen, liver and hollow viscera is not uncommonly seen in the presence of no signs of external injury.

It is well known that in children or young adults, compression of the chest often occurs with extensive internal injuries. On examination, due to the flexible nature of the ribs, no fracture may be found, and often no signs of external violence.

In some cases the injuries sustained, though fatal, may have been caused by the falling of the body against the pavement or curb, the actual contact with the vehicle being slight. A careful and thorough examination cannot be overstressed, since we are all but too familiar with the endeavors of some ubiquitous and wrangling lawyers to defeat, *ora e sempre*, the ends of justice. It must be remembered that in court, before a lay jury, testimony proving that the car passed over the victim (or struck him with great violence) carries more weight in the final issue of the case.

It is my experience that only in a small group of cases does the body show marks sufficiently characteristic to denote actual

contact with an automobile. The importance of these marks, when present, even though at times slight, is self evident. In

Fig. 1. Automobile accident; pedestrian struck by truck. Brush abrasions, linear in character, across lower right abdominal quadrant, running parallel with Poupart's ligament, caused by tire of truck. Compression of abdomen, rupture of mesentery of small intestine, intra-abdominal hemorrhage.

Fig. 3. Automobile accident; pedestrian struck by pleasure car. Brush abrasions over outer surface of right arm and forearm produced by radiator. Multiple fractures of ribs, compression of chest, bilateral hemothorax.

rare instances, such conclusions may help in the prosecution of the "hit and run" driver.

Such characteristic marks, indicating actual contact of the body with certain parts of the car, may take innumerable forms. I cite but a few: Abrasions, often in parallel linear form, are so characteristic that the passing of a tire across the body is unquestionable (Figs. 1, 2). Abrasions or skin bruises which are honeycombed or parallel, leave no doubt as to the imprint of the radiator (Fig. 3). We see also de-

Fig. 2. Automobile accident; run over by rear wheel of heavy sedan. Tire imprint across right side of neck and face. Multiple fractures of ribs, compression of thorax and fracture of fourth, fifth and sixth cervical vertebrae.

Fig. 4. Automobile accident; pedestrian struck by "hit and run" driver. Entire radiator cap found inside chest. Note bottom of cap showing through wound in chest. Compression of chest, multiple fractures of ribs and sternum, rupture of heart and lungs. Entire cap shown in insert. (Figs. 1–4 from Office of Chief Medical Examiner, Essex Co., N. J.)

pressed or punched fractures of the skull from projecting radiator caps, puncture wounds of the chest caused by figures used to adorn the modern radiator (Fig. 4), glass embedded in the body from a broken windshield.

Lastly, as many automobile victims live several days before dying, hospital records should contain accurate descriptions of all injuries and circumstances under which the patient was admitted.

# PUERPERAL INFECTION

## WITH SPECIAL REFERENCE TO THE IMPORTANCE OF ANATOMICAL PATHWAYS IN THE SPREADING OF PYOGENIC INFECTION

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### SIMILARITY BETWEEN PUERPERAL INFECTION AND ANY OTHER INFECTED WOUND

INFECTION of the uterus following childbirth and abortions is a wound infection identical, in most respects, with any other infected wound; the differences, in the main, being the type of tissues infected and the enormous venous and lymphatic drainage from the child-bearing organs.

Puerperal infection is the result of bacterial invasion of gross or microscopic puerperal wounds of the uterine cavity or cervix and occasionally the vagina and perineum.

The infection may remain localized, or it may spread to other parts of the body as the result of drainage along the tissue interstices, lymphatics, blood vessels, or by continuity through patent lumina (fallopian tubes).

### PUERPERAL INFECTIONS OF GREAT INTEREST TO PATHOLOGISTS AND MEDICAL EXAMINERS

Of all cases coming to autopsy either in a medical examiner's office or on the necropsy tables of general hospitals, none are so interesting to the morbid anatomist as those in which a definite logical sequence of pathological events can be established at the postmortem table.

Uterine infections are outstanding in this respect, since the correct interpretation of what occurs is usually clear and demonstrable to the naked eye.

In addition, puerperal infections are of interest to medical examiners since all deaths from suspected criminal abortion and most deaths from spontaneous, accidental and therapeutic abortion must be investigated by them. Those dying from puerperal sepsis following full term deliveries, however, do not so often come to our attention, since there is usually no suspicion of anything criminal in such cases. However, these two types of infection, regardless of time onset, are identical.

### IMPORTANT PHYSIOLOGICAL DATA

The vagina during pregnancy contains a bacterial flora of considerable variety, containing chiefly varying strains of aerobic streptococci and staphylococci, B. coli communis, B. welchii, and saprophytes (consisting mainly of anaerobic streptococci).

Normally the secretions of the vagina and cervix check the growth of these organisms.

However, after but six hours of dry labor many of these organisms can be found in the cervix and lower uterine segment. In such cases, ordinarily, the local tissue reactions are such that in the course of seventy-two to ninety-six hours, these same bacteria disappear without doing any damage.

After labor the firm contractions and retraction of the uterus obstructs the venous return through the terminal radicals in the basal endometrium. These sinuses become engorged, and if contraction is maintained, serum escapes and cells migrate into the surrounding stroma. This irritation causes a rapid proliferation of small round cells and a definite subendothelial cellular defense barrier is formed. Owing to the blood supply being cut off a superficial necrosis of the thickened endometrial lining takes place and is cast off piecemeal in the lochial discharge.

In addition, a variety of microorganisms, not normal inhabitants of the vagina, may be mechanically introduced from without, by instrument or by the operator's hand, either in performing a dirty abortion or by too frequent vaginal examinations, instrumentation, etc., during labor.

There is considerable argument as to whether puerperal infections are caused by microorganisms already present in the genital tract or by those introduced from without. Certainly, the most frequent and dangerous of puerperal infections are caused by hemolytic streptococci (beta). They are probably almost always exogenous invaders.

## PRIMARY LESIONS IN PUERPERAL INFECTION

*Puerperal Ulcer:* The so-called puerperal ulcer is the simplest type of puerperal wound infection. Such ulcers were of common occurrence before the introduction of aseptic obstetrical methods. They are infequent at the present time.

This ulcer is caused by bacterial inoculation of the surfaces of tears about the vulva and perineum. A similar infection, however, may occur on cervical tears.

As a rule, the puerperal ulcer gives rise to very little systemic disturbance. It is surrounded by an inflammatory defense barrier which is usually sufficient to limit the infection.

*Cervical Tears:* When the infected tear extends into the adjacent cellular tissues around the cervix and vaginal vault, a protective parametritis develops.

If the resistance of the host, however, has been reduced by excessive trauma or loss of blood, or the virulence of the bacteria is great, these protective barriers may be weakened and infection reach the lymphatics and blood stream with disastrous results.

*Puerperal Endometritis:* A septic endometritis is the real starting point of nearly all fatal puerperal infections. It is the primary lesion; the endometrium being the portal of entry of the invading organisms.

When the uterus fails either to contract and retract properly, or its contraction is not maintained (subinvolution) or where the infecting organisms are of great virulence and dosage, the perfect physiological defense mechanism is broken down and the placental site, or the entire interior of the uterus, may become infected.

The superficial necrotic tissues cut off from their blood supply and left adherent to the uterine wall, the detached and dead pieces of placenta stagnating in the uterine cavity, offers the best possible culture medium for hemolytic streptococci and other organisms. Rapid growth takes place until the organisms, greatly increased in numbers and virulence, attack victoriously the uterine wall.

Drainage through the lymphatics to the peritoneum or to the regional nodes, blood stream and infection of the thrombosed venous sinuses may then rapidly take place with all the secondary lesions seen in puerperal sepsis.

## CAUSE OF DEATH IN PEURPERAL INFECTION

In 105 consecutive autopsies on cases of suspected criminal abortion I have found the causes of death as shown in Table 1:

TABLE 1

CAUSES OF DEATH IN 105 AUTOPSIES ON SUSPECTED CRIMINAL ABORTION (MARTLAND)

| | Cases | Per Cent |
|---|---|---|
| 1. Peritonitis | 53 | 50.4 |
| 2. Thrombophlebitis | 33 | 31.4 |
| 3. Putrid endometritis (gas anaerobes) | 8 | 7.6 |
| 4. Uterine erysipelas (Sepsis foudroyant, acute lymphatic sepsis) | 0 | 0.0 |
| 5. Hemorrhage | 10 | 9.5 |
| 6. Air embolism | 1 | 0.9 |

It will be noted that over one half of the cases showed peritonitis and about one-third septic thrombophlebitis, the two conditions causing over 80 per cent of the deaths.

The incidence of these main types of infection is practically identical in post-abortive and puerperal sepsis occurring after the later stages of pregnancy.

Kneise[1] in 89 autopsies on puerperal infections found the frequency of the various lesions to be as shown in Table II:

TABLE II
CAUSES OF DEATH FROM PUERPERAL INFECTION IN 89 AUTOPSIES (KNEISE)

| | Cases | Per Cent |
|---|---|---|
| 1. Peritonitis | 43 | 48.3 |
| 2. Thrombophlebitis | 20 | 22.5 |
| 3. Pyemia | 17 | 19.1 |
| 4. Parametritis | 7 | 7.9 |
| 5. Sepsis foudroyant | 2 | 2.2 |

It will be noted that the incidence of peritonitis and thrombophlebitis is practically the same in both tables, and form from 70 to 80 per cent of the fatal cases.

In addition, the cases classified as pyemia by Kneise might be added to his cases of thrombophlebitis since, in my experience, practically all cases of pyemia show evidence of an active or healing thrombophlebitis.

### ACUTE PUERPERAL PERITONITIS

A generalized, rarely localized, peritonitis is the common cause of death in puerperal infection and septic abortion, forming approximately 50 per cent of the fatal cases.

CLINICAL COURSE. The clinical signs, symptoms and treatment do not differ from those of other types of acute general peritonitis. Puerperal peritonitis is almost hopeless and death usually occurs within ten days after delivery. The infection has nearly always spread over most of the peritoneal cavity.

AUTOPSY FINDINGS. In peritonitis cases the following conditions may be found:

*Puerperal Endometritis.* The interior of the uterus in most cases is quite dirty, containing a sloughing mass of necrotic material, decidual debris, foul blood clots, etc. The infection spreads over most of the uterine cavity and is not limited to the placental site, as in the cases of thrombophlebitis. Ulcerative areas may be covered with a pseudo-diphtheritic membrane.

Microscopic examination shows the necrotic lining to be teeming with organisms. Beneath is a poorly developed zone of leucocytic defense; however, it may be entirely lacking and bacteria may be seen extending by way of the lymphatics into the deeper layers of the decidua, muscularis and making their way towards the peritoneum.

A strong defense reaction is often seen in the muscularis; namely, the phagocytosis of organisms by numerous clasmatocytes, large cells belonging to the reticulo-endothelial system with small nuclei and vacuolated cytoplasm, situated in areas of loose, juvenile connective tissue (defense reaction of Hofbauer).

In some cases, when the organisms are virulent hemolytic streptococci, the signs of an endometritis may be very slight and the interior of the uterus clean, brawny and edematous. If uterine erysipelas or sepsis foudroyant does not occur, the infection passes rapidly through the uterine lymphatics and extends to the peritoneum causing peritonitis.

*Metritis.* The underlying muscularis is involved in most cases. It may be only slight with small scattered areas of leucocytes, or there may be definite abscess formation.

In many cases, multiple abscesses occur. When present, they are chiefly situated beneath the peritoneum, since the lymphatic network is greater in this area. The so-called *subserosal abscess* is one of the most frequent causes of the subsequent peritonitis, the peritoneum being infected by contiguity or by actual rupture of the lesion.

Sometimes, a considerable portion of the muscularis may become involved and, leading to necrosis, be expelled as a large cast, the so-called metritis dissecans or gangrenous endometritis.

*Extension of Infection Direct to Peritoneum by Lymphatics of Uterine Wall with Production of Peritonitis.* Organisms, especially hemolytic streptococci, may rapidly make their way, by means of the lymphatics from the interior of the uterus, to the peritoneal surface and give rise to a

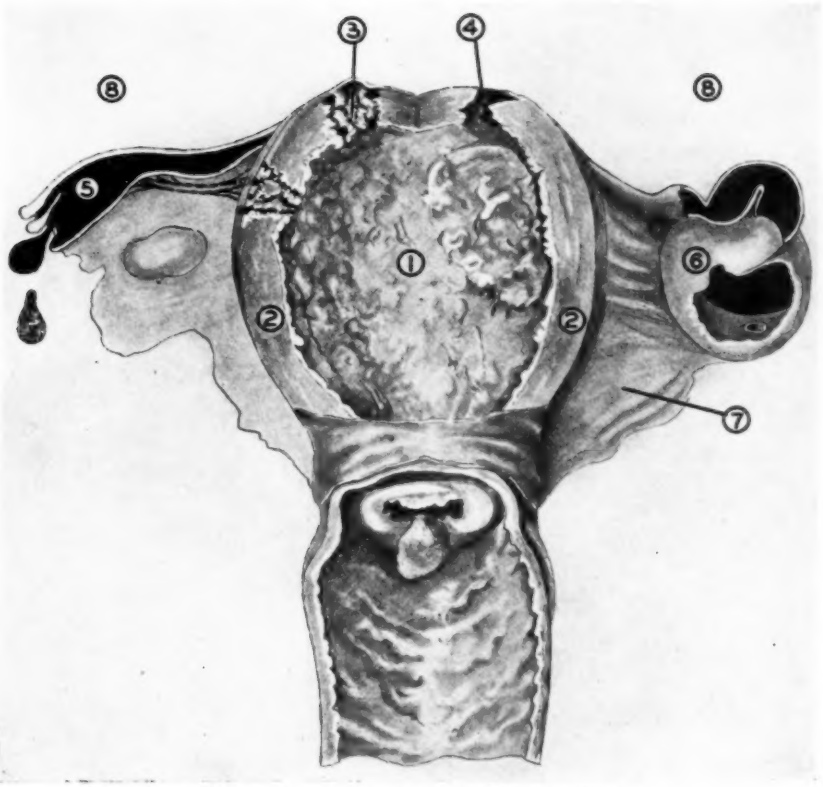


FIG. 1. ACUTE PUERPERAL PERITONITIS.

*Summary of Lesions*

1. Puerperal Endometritis
   Septic endometritis the primary lesion
   Occasionally a puerperal ulcer.
2. Metritis
   The underlying muscle may show only microscopic involvement, or there may be multiple abscesses or a gangrenous endometritis.
3. Extension of Infection Direct to Peritoneum by Lymphatics of Uterine Wall with Production of Peritonitis
   This is probably the most common way in which the peritoneum becomes infected, the subserosal abscess being an important factor.
4. Extension of Infection Direct to the Peritoneum by Uterine Trauma induced by Instrumentation
   Rupture of uterine wall caused by instrumentation.
5. Extension of Infection to Peritoneum by Continuity through Lumina of Fallopian Tubes
   While such authorities as Williams and MacCallum state that this does not occur, the writer firmly believes that this is a very important pathway. See text.
6. Production of a Salpingitis or Tubo-Ovarian Abscess with Localized Peritonitis
7. Extension of Infection to Pelvic Cellular Tissues
   With formation of paramatritis, pelvic abscess, etc.
8. Peritonitis
   Localized and generalized.

FIG. 2. THROMBOPHLEBITIS.

*Summary of Lesions Due to Infection of Veins*

1. Infection of Endometrium at Placental Site
   Two different cases are illustrated in the figure.
   On the right side (left side of figure) the infection is situated in upper uterine zone. In this location, the thrombophlebitis is more apt to extend to the ovarian vein.
   On the left side (right side of figure) the infection is in lower uterine segment and extends to uterine vein.
2. Thrombophlebitis of Uterine Wall
   Note localized and unilateral character.
3. Thrombophlebitis in Broad Ligament
   Note unilateral location.
4. Extension of Thrombophlebitis to Ovarian Vein
   Note unilateral involvement only.

4′. Extension of Thrombophlebitis to Uterine and Internal Iliac Veins

5. Periphlebitis of the Ovarian Vein—Retroperitoneal Abscess

5′. Production of Pelvic Cellulitis and Retroperitoneal and Extraperitoneal Abscess

6. Phlegmasia Alba Dolens
   Due to retrograde venous infection.
   For other causes, see text.
7. Infection Carried to Right Side of Heart
8. Acute Bacterial Endocarditis of Tricuspid or Pulmonic Valves
   This is an unusual occurrence.
9. Paradoxical Embolism
   Emboli to systemic arterial circulation through a patent foramen ovale.
10. Embolic, Hematogenous Abscesses of Lungs
    This is the most important and characteristic secondary lesion of thrombophlebitis cases, whatever their cause.
11. Subpleural Abscess Infecting Pleura by Contiguity or Rupture
12. Empyema
    From infection by close proximity of lung abscess.

*No peritonitis in these cases.*

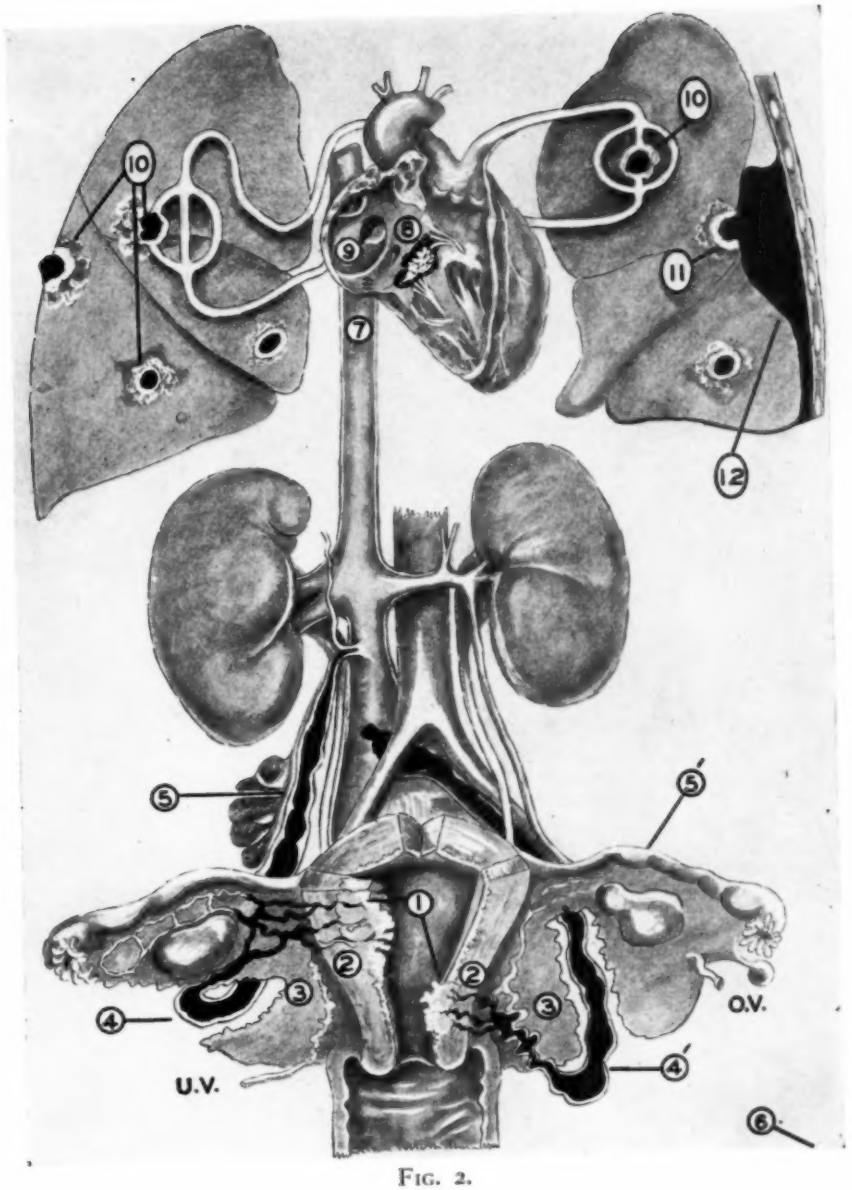


FIG. 2.

FIG. 3. THROMBOPHLEBITIS.

*Summary of Lesions Due to Infection of the Arterial Circulation*

1. Organisms enter Left Auricle via Pulmonary Veins
2. Acute Bacterial Endocarditis of the Mitral or Aortic Valves
3. Emboli to Myocardium
   Via coronary circulation. Microscopic if due to streptococci. Distinct naked eye abscesses if due to staphylococci.
4. Extension of Infection to Pericardium
   Subpericardial abscess infecting pericardium by contiguity or rupture with production of a serofibrinous pericarditis, or empyema of pericardium.
5. Emboli to Nervous System
   With production of petechial hemorrhages due to septic emboli; large cerebral hemorrhages due to septic thrombosis of larger vessels; multiple, usually small, brain abscesses and suppurative meningitis.
6. Splenic Infarcts
   Typical wedge-shaped infarcts or purulent abscesses. Localized perisplenitis and rarely perisplenic abscess.
7. Renal Infarcts
   Isolated infarcts; suppurative embolic nephritis; perinephric abscess.
8. Cutaneous and Mucous Membrane Emboli
   In conjunctivae, skin and mucous membranes. At autopsy in pleura, pericardium and peritoneum. Raised liquefied centers in staphylococcus infections.
9. Embolic Lesions in Eyes
   Retinal petechiae and rarely panophthalmitis.
10. Embolic Lesions in Joints
    Suppurative arthritis most common in shoulders, elbows and knees. Pyemia.
11. Osteomyelitis
12. Embolic Lesions in Arteries
    Arterial occlusion in peripheral arteries with gangrene. Mycotic aneurysms.

    Many of these lesions often keep up the sepsis after the original source has entirely healed and disappeared. Even secondary and tertiary lesions may act as primary ones, many months later.

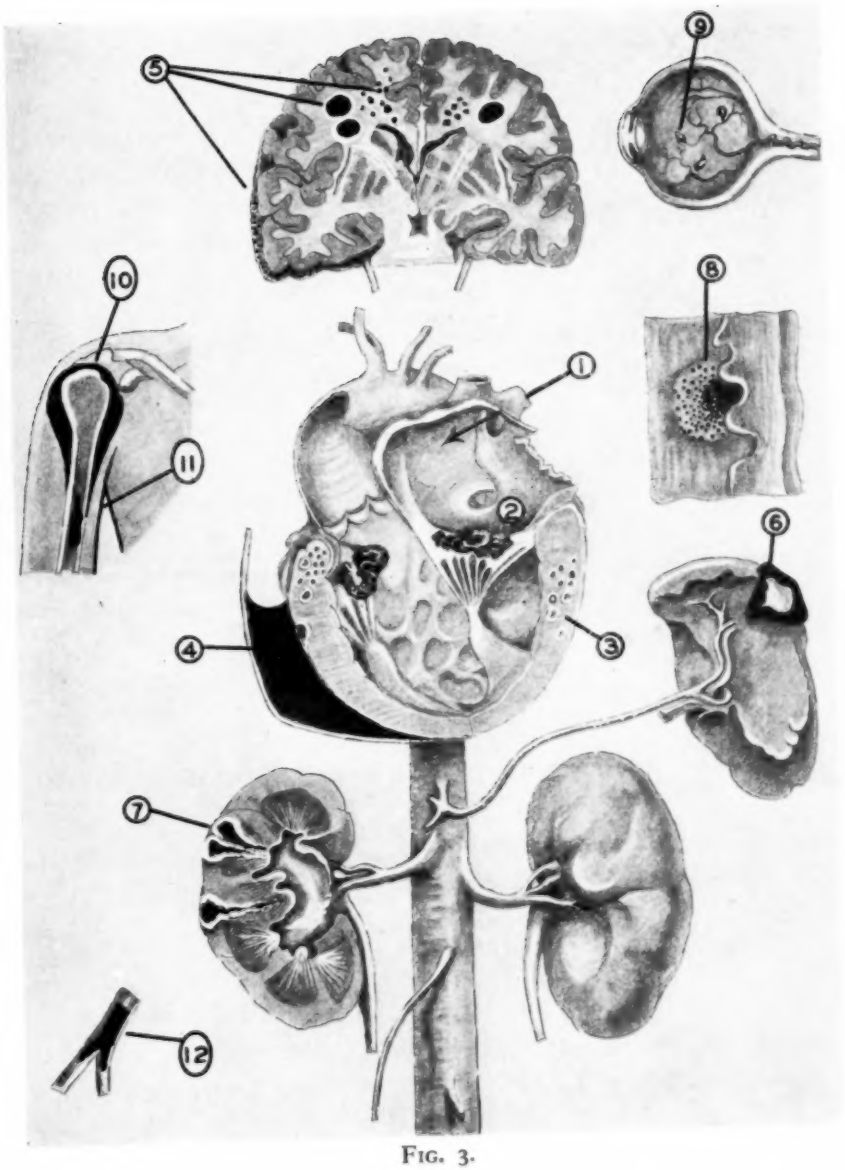


FIG. 3.

FIG. 4. UTERINE ERYSIPELAS.

Acute Lymphatic Sepsis (Tweedy and Wrench); Sepsis foudroyant (Williams et al.).

*Summary of Lesions*

1. Infection of Endometrium with Streptococci
   The uterus is slightly enlarged, *clean*, boggy, red and edematous. Its endometrium is smooth and glistening.
2. Drainage into Lymphatics of Uterine Wall
3. Drainage into Lymphatics of Broad Ligaments
   The process is bilateral, uniform and symmetrical.
4. Drainage into Regional Lymph Nodes of Pelvis and Pelvic Brim
   The glands are swollen, edematous, pale and distended with watery fluid containing innumerable streptococci.
5. Drainage into Lumbar Lymph Nodes and Thoracic Duct
   Rapid death, from toxemia, results in nearly all cases.
6. Drainage into Left Subclavian Vein
   Patients usually die before this occurs.
7. Other Lesions Due to Toxemia
   Acute cloudy swelling of heart muscle, liver and kidneys, and acute septic spleen. *No emboli and no peritonitis in these cases.*

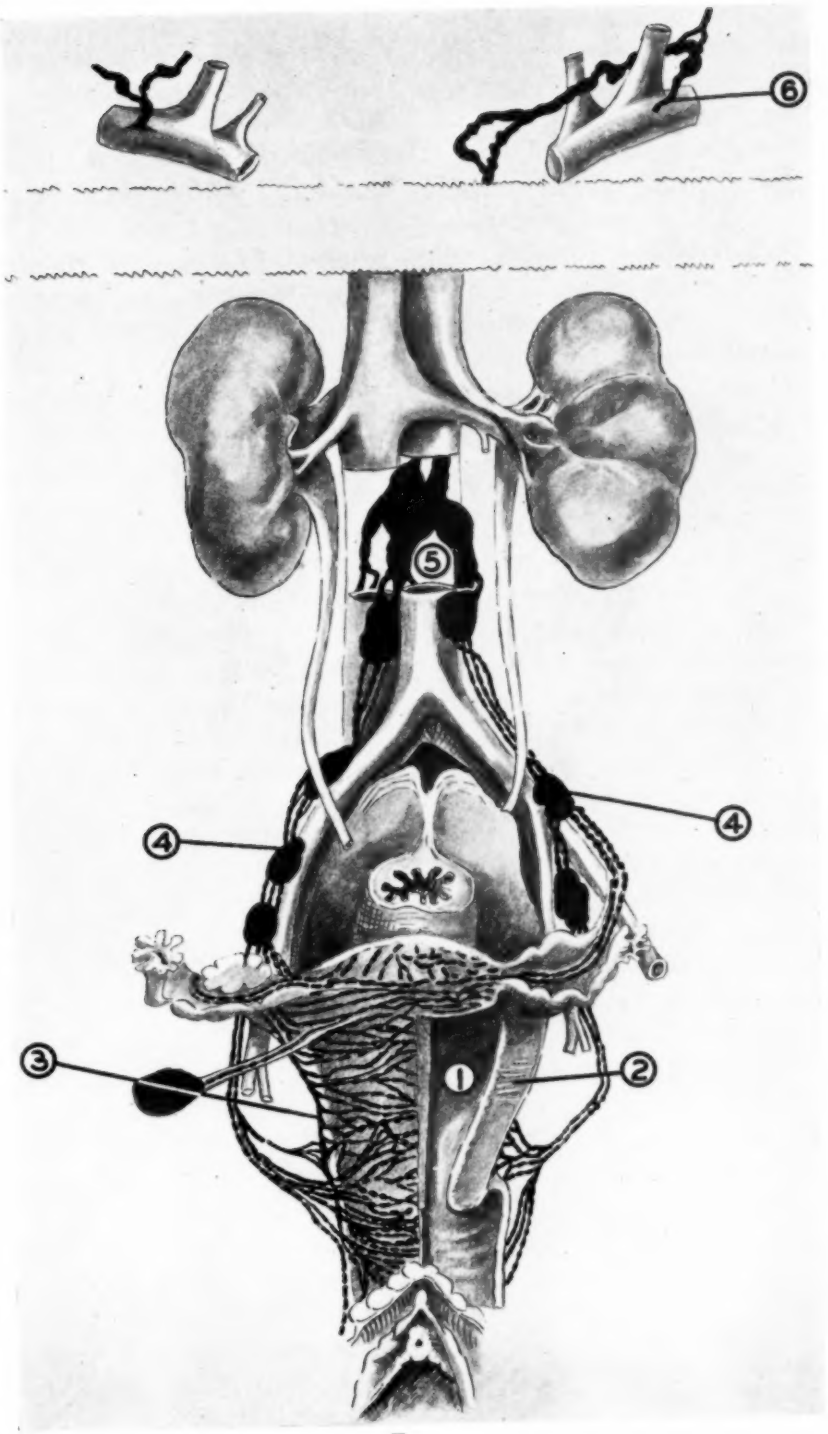


FIG. 4.

peritonitis with or without the production of subserosal abscesses.

Williams thinks this is the usual way in which peritonitis develops in these cases. This direct extension to the peritoneum may be only recognizable on microscopic examination, seen as small localized areas, or, on the other hand, a considerable portion of the uterine wall may show perilymphangitis with extensive leucocytic infiltration, and chains of streptococci filling the lymphatic lumina and making their way to the peritoneum.

*Extension of Infection Direct to the Peritoneum by Uterine Trauma Induced by Instrumentation.* It is obvious that if the uterine wall has been mechanically traumatized by instruments in performing, for instance, an abortion, a more or less direct communication with the peritoneum may be established; due to a solution in continuity of the uterine wall by such a trauma, the currettage by the abortionist of the pelvic contents, with partial evisceration, is not unheard of.

In this respect it should be noted that a single instrumentation often does little harm, the danger lying in the second introduction which stirs up the bacteria originally introduced.

*Extension of Infection to Peritoneum by Continuity through Lumina of Fallopian Tubes.* It is the opinion of many recent writers that a direct extension from the uterine cavity to the peritoneum, by way of the lumina of the fallopian tubes, does not occur in puerperal or postabortive infections.

Williams[2] states:

> The great majority of writers will tell you that in many cases the infection is conveyed from the uterus to the peritoneum by means of the tubes; yet in the many autopsies which I have seen upon women dead from puerperal infection, I have never seen one in which there was any evidence that infection has occurred in this manner.

He was of the opinion that it was exceptional for infection to extend from the uterine cavity to the fallopian tubes, and that when it did occur, it was often only an extension of the process in the uterus, by way of the lymphatics into the outer walls of the tubes; further, that while the folds of the tubal mucosa may even contain leucocytes and streptococci, the lumen is free from infectious material.

Furthermore, in those cases showing involvement, the lesions are more marked at their fimbriated ends indicating a secondary involvement from the peritoneum, a perisalpingitis with invasion finally of the endosalpinx, the tubes being attacked from without.

MacCallum,[3] speaking of peritoneal involvement in puerperal sepsis, states: "The fallopian tubes play no part in this."

It has been my experience, however, especially in postabortive sepsis, that the peritoneum is frequently infected by this route. It has not been uncommon in cases of streptococcal salpingitis to find at autopsy thin pus leaking from the fimbriated extremities, the tubes themselves containing pus as far as the uterine isthmus. In 105 cases of abortion examined by me, in which 53 women died as the result of peritonitis, 28 cases showed bilateral and 11 cases a unilateral suppurative salpingitis. Histological examination of many of these tubes show the main lesions to be in the mucosa and submucosa.

Furthermore, the following well-known facts may be cited to demonstrate the logic of such a pathway:

It is the concensus of opinion that in gonorrheal salpingitis the infection is a mucosal one traveling directly from the uterine cavity into the tubes. In old cases, of course, both ends become sealed and a pyosalpinx results.

At laparotomy, retrograde menstrual flow of blood escaping from the fimbriated ends of the tubes has often been seen. Sampson[4] is of the opinion that gonococci are often carried into the tubes with such a retrograde flow.

The drainage of pus from the dependent distal ends of the tubes in a gonorrheal

salpingitis is the most plausible explanation of a tubo-ovarian abscess, the bacteria directly entering a recently ruptured follicle or corpus luteum.

In endometriosis, endometrial tissue composed of cellular elements of appreciable size undoubtedly, in some cases, reach the peritoneum by way of the lumina of the tubes, and implanting themselves on ovaries and pelvic tissues, produce chocolate cysts and pelvic adenomata.

Furthermore, the patency of the tubes as an anatomical pathway is easily demonstrated by the ease in which air reaches the peritoneal cavity in the performance of the Reuben test.

Why then must the lumina be closed only in puerperal sepsis?

Production of a Salpingitis or Tubo-ovarian Abscess with Localized Peritonitis. The infection of the tubes just cited may, in some cases, last long enough to seal off both ends of the fallopian tubes with a resultant suppurative salpingitis and pyosalpinx. Often, the ovaries are enlarged to many times their normal size and markedly edematous.

As even the normal fallopian tube has its fimbriated extremity in a dependent position, pus may more easily run by gravity out of such a pus tube and directly contaminate the ovary, the infection entering a recently ruptured follicle or corpus luteum. The whole process is enclosed in a localized peritonitis, and due to excavation and distention, a large abscess cavity is formed in the region of the ovary.

Extension of Infection to Pelvic Cellular Tissues. The pelvic cellular tissues may become infected either from the adjacent salpingo-öophoritis and inflammation of the pelvic peritoneum (perimetritis), or by direct extension through the uterine wall (parametritis). Finally, especially in cervical tears, a direct infection of the pelvic cellular tissue may occur. The tissues may be only edematous or the inflammatory edema may progress to abscess formation. Pelvic abscesses often are large and may spread anteriorly dissecting off the peritoneum from the anterior pelvic and abdominal wall, and eventually point over Poupart's ligament. However, they may spread posteriorly and form large retroperitoneal phlegmons (infections with pus formation in loose cellular tissues).

The inflammation may extend to the tissues of the thigh and form an unusual type of phlegmasia alba dolens, a periphlebitis via the lymphatics.

*Peritonitis.* In most cases the peritonitis, which eventually kills the patient, is often localized at first due to the formation of abscesses in the posterior cul-de-sac which are walled off at the pelvic brim by the sealing effects of the great omentum. Often, marked attempts of nature to limit the infection to the pelvic cavity may be seen. A general infection of the peritoneum, however, usually results.

## THROMBOPHLEBITIS

Septic thrombophlebitis is the second most common form of death in puerperal infections, being exceeded only by those of peritonitis, and forming from 30 to 40 per cent of the fatal cases.

In this type of puerperal infection, the process originates at the placental site. During the removal of a retained or adherent placenta, local infection may occur, or a piece of placenta, left behind, may form a favorable medium for streptococci and staphylococci. The abundant venous sinuses at the placental site which have become occluded by blood clots form an ideal medium for bacterial growth.

The infection spreads outward through the veins of the uterine wall, with resulting thromboses. This process extends beyond the uterus into the veins of the broad ligament and thence to the common iliac, ovarian veins and even the vena cava.

Clinical Course. Symptoms do not occur, as a rule, until the end of the first or the beginning of the second week, since it requires time for the bacteria to loosen the clots by peptonization.

The onset is usually sudden. There is a chill. The teeth chatter, the lips become blue and the face is drawn and pinched. Both temperature and pulse-rate rise, the former reaching 104 to 105°F. This is followed by a profuse perspiration, the temperature and pulse fall to *normal*, and the patient feels quite well.

A few hours later or the next day, a similar fastigium is reached, and the course now becomes one characterized by chills, high fever, profuse sweating and remissions recurring daily in succession (spiked temperature chart).

With the establishment of a suppurative thrombophlebitis and subsequent feeding of the venous circulation with septic clots containing organisms, there is hardly any portion of the body which cannot become infected.

The feeding of the blood stream may be more or less continuous if there is a constant breaking off of emboli. It occurs in showers in most cases. A condition of septicemia or bacteremia is said to exist,—the recognition of bacteria in the blood. As bacteria do not multiply in the blood, save in some cases where death is approaching, a continuous or spasmodic shower into the blood from primary or secondary suppurating lesions is the only reasonable explanation.

As the disease progresses, the interval between the rigors becomes shorter and both temperature and pulse-rates become permanently raised, the patient becoming weaker and finally dying. Recovery may occasionally take place. In fatal cases the patients usually live five to six weeks or longer.

In other cases, the course may become more chronic, especially when pyemia develops, and may be several months in duration with either final recovery or death. Although the disease is frequently fatal many desperate patients eventually recover. It is never safe to give a good prognosis as sudden serious complications such as endocarditis, meningitis, etc., may arise at any time. Recovery may be complete, or the patient may be a chronic invalid from arthritis, frozen pelvis, etc.

Autopsy Findings. In autopsies on such cases, the following pathological events in more or less chronological order, may be encountered:

### *Lesions Due to Infection of Veins*

*Infection of Endometrium at Placental Site.* The infection of the endometrium occurs at the placental site. Due to this localization, since the process only rarely extends over the interior of the uterus, the venous drainage is unilateral. The infected area is shaggy, necrotic and purulent.

Likewise, it is important to note the location of the endometritis. If it is in the upper uterine zone, an extension of the thrombophlebitis to the ovarian vein is more apt to occur than if the placental site is low down. Drainage from the lower portion of the uterus is more apt to take place into the uterine and internal iliac veins.

Microscopic sections show an extensive, necrotic and suppurating endometritis with placental shadows, decidual cells, etc., upon which a positive diagnosis of pregnancy can usually be made.

*Thrombophlebitis of Uterine Wall.* In most cases there can be seen, with the naked eye, definite thrombosis of the veins in the uterine wall directly beneath the infected endometrium. Microscopic study is conclusive.

*Thrombophlebitis in Broad Ligament.* Drainage takes place into the veins of the broad ligament practically always on one side, since the lesion is usually unilateral. The veins may be extensively thrombosed with fairly clean or purulent clots. Due to marked edema and thickening of the broad ligament, it may be felt clinically and is often tender.

In other cases, a honeycombing of the tissues of the broad ligament may be seen with multiple small abscesses. A generalized peritonitis is never seen in pure, uncomplicated types of thrombophlebitis.

*Extension of Thrombophlebitis to Ovarian Vein.* In many cases, especially when the placental site is high, as it usually is, there is an extension of the thrombophlebitis into the ovarian vein.

The vein involved is always unilateral and it may be distinctly seen as a distended cord with a fairly clean but usually purulent clot. It may reach as high as the opening into the inferior vena cava, or there may be a separate intimal lesion in the vena cava.

Often, especially when the patient has lived for some time, the top of the clot may be firmly adherent and entirely block the lumen of the vein; in consequence, there is a mechanical prevention of agglutinated bacteria, leucocytes, fibrin, etc., from entering the venous blood stream. This is an attempt of nature to cure the infection, and apparently has nothing to do with any immunological process. The vein in many such cases may be distended with pus below the point of closure.

In still other cases, the process may subside and convert the ovarian vein into a thickened cord; death taking place from metastatic foci.

Likewise, the uterus may return entirely to a normal state, making, at times, the question of an abortion an extremely difficult one to prove.

*Extension of Thrombophlebitis to Uterine and Internal Iliac Veins.* In many cases, especially when the infection of the endometrium is in the lower portion of the uterus, identical lesions, which are nearly always unilateral, may be seen in the uterine vein. The further extension of the process into the internal iliac vein and vena cava is not uncommon.

The sealing-off of these venous channels often accounts for negative blood cultures in these cases. What is more important, however, is the purification of the venous blood by the capillary beds of the lungs.

*Periphlebitis of the Ovarian Vein; Retroperitoneal Abscess.* Often a periphlebitis of the ovarian vein may be encountered where the walls of the vessels, perivascular lymphatics and surrounding tissues are infiltrated with leucocytes. Pockets of pus may occur in the surrounding tissues, and often, large collections, occurring retroperitoneally over the psoas muscle, may sometimes be seen due to an infection by contiguity. On account of the loose cellular tissues in this region, such abscesses are often of the nature of a phlegmon (there is no definite, circumscribed accumulation of pus).

*Production of Pelvic Cellulitis and Retroperitoneal and Extraperitoneal Abscess.* In a manner similar to the formation of retroperitoneal phlegmons over the psoas muscles, a periphlebitis of the uterine and internal iliac veins may cause collections of pus in the lateral walls of the pelvis, unilateral, usually, in distribution.

*Phlegmasia Alba Dolens.* A retrograde embolism into the femoral vein may sometimes take place with the production of a phlegmasia alba dolens. The process is entirely septic in nature.

On the other hand, the thrombosis in the femoral vein may be entirely static and the clot aseptic (static thrombosis of Aschoff). The danger of sudden death from pulmonary embolism in such cases is well known.

Again, in rare instances, the vein may be thrombosed by a periphlebitis due to lymphatic drainage from the pelvis into Scarpa's triangle.

*Infection Carried to Right Side of Heart.* When there is a septic, suppurating clot either in the veins of the broad ligament, in the ovarian or uterine veins, in the internal iliac vein or inferior vena cava, bacteria in a finely divided form, or usually larger pieces of agglutinated cocci, clinging masses of leucocytes, blood platelets and fibrin, are discharged directly into the venous circulation and carried to the right side of the heart. This discharge is usually intermittent but occasionally more or less continuous.

Sometimes the discharge at intervals of a considerable amount of pus (teaspoonful) which has been locked up in some

vein may give an exceptionally high leucocytosis.

*Acute Bacterial Endocarditis of Tricuspid or Pulmonary Valves.* Infection of the right side of the heart is unusual, as both tricuspid and pulmonary valves are infrequently the seat of extensive rheumatic lesions (so often the antecedent of an acute or subacute bacterial endocarditis).

When right-sided endocarditis occurs the offending organisms are more likely to be pneumococci or gonococci.

*Paradoxical Embolism.* Paradoxical or crossed embolism denotes a systemic arterial embolism, produced from particles liberated in the veins which have avoided the lungs by means of a short circuit through a patent foramen ovale.

The foramen ovale is patent in at least 20 per cent of all adults, but the opening is usually only a small slit in its anterior portion and is physiologically closed by the greater pressure in the left auricle. While probably of unusual occurrence there is always the possibility of septic emboli passing through such an opening directly into the left side of the heart.

Paradoxical fat emboli lodging in the brain are more common, since in trauma with extensive bone injury, a condition of surgical shock often exists allowing the pressure in the right auricle to approach that of the left with the resultant opening of the physiologically closed but patent foramen.

*Embolic, Hematogenous Abscesses of Lungs.* Once the emboli and agglutinated bacteria reach the right side of the heart, the great defense barrier of the venous system comes into play; namely, the lungs.

The lungs are the most important and extensive capillary filtering bed of the venous circulation for large particles.

In cases of septic thrombophlebitis, constant showers of hemolytic streptococci or staphylococci, mixed with blood platelets, fibrin, and polymorphonuclear leucocytes pass rapidly through the right side of the heart and enter the lungs. Most of the organisms are filtered out, or retained in the lungs and multiple, symmetrical, bilateral, hematogenous, embolic abscesses result. This enormous filtering capacity of the lungs most readily explains the negative blood cultures often obtained in these cases (using standard blood culture technique*).

On the other hand, when the bacteria are finely divided and the size of the embolic masses is very small, they may pass through the lungs with a resultant systemic infection. In this respect it is of interest to note that in the bacteremia of secondary syphilis, the spirochetes are unaccompanied by large particles, such as agglutinated bacteria, etc., and it is reasonable to assume that most of the organisms pass through the lung capillaries and enter the arterial circulation. For this reason syphilis of the lung is exceedingly rare.

The appearance of embolic abscesses in the lungs is very characteristic, and, when seen, always denotes a septic thrombophlebitis elsewhere in the body.

The abscesses may range in number from 2 or 3 to 15 or 20, or may be innumerable, and in size from small discrete, though sometimes confluent, lesions either just visible as small reddish areas of consolidation 1–2 mm. in diameter to typical abscesses 1 or 1.5 cm. or more in diameter.

Their centers are grayish or reddish gray in color, sometimes yellow, and liquefaction takes place rapidly, especially in staphylococcal infections, forming small abscesses surrounded by a dark red border of hemorrhagic pneumonic consolidation, outside of which the lung is edematous. The intervening lung is usually free.

Microscopically in small lesions, which do not show extensive liquefaction, colo-

* By Standard Technique is meant the use of media that have stood the test of time, in contradistinction to the methods employed by imaginative arthritis experts in isolating a single streptococcus from the blood in apical root abscesses, rheumatism, arthritis deformans and conditions that could be named ad infinitum.

It is obvious that such delicate methods, even if free from the suspicion of contamination caused by too frequent opening and long incubation, are useless for either diagnosis or prognosis in puerperal infections.

nies of cocci lodged in the lumen of a central blood vessel may be seen.

Those near the visceral pleura often bulge externally, their liquefied centers making them appear like small subpleural abscesses, which truly they are.

The pleura over such lesions is often dull, lustreless, and covered with a fresh fibrinous exudate.

*Subpleural Abscess Infecting Pleura by Contiguity or Rupture.* Once the infection has reached the periphery of the lung with the formation of subpleural abscesses, the danger of pleural infection is great.

Streptococci can pass through the intact pleural barrier, by either continuity (being phagocyted by wandering reticulo-endothelial cells), through microscopic areas of the broken or liquefied pleural barrier, or a subpleural lesion may rupture directly into the pleural cavity. Frequently, at autopsy, a blown-out hole may be seen in the visceral pleura over the center of one of these abscesses. Such an occurrence is rarely the cause of the empyema, as it usually is a terminal event, the empyema often antedating it by several days.

*Empyema.* A characteristic, usually streptococcic, empyema is thus produced. It is often bilateral, since the infection is equally distributed over both lungs.

The fluid, most often watery and more serous than purulent, rapidly accumulates and slowly changes to a characteristic thin pus, taking usually two to three weeks to become frankly purulent. It contains usually numerous streptococci or other organisms, disintegrating pus cells with little or no phagocytosis.

Adhesions and encapsulation are slow and most often do not take place at all. The treatment is that of any streptococcal empyema, aspiration being safer than rib resection, since the mediastinal partition is not fixed.

### *Lesions Due to Infection of the Arterial Circulation*

*Organisms Enter Left Auricle via Pulmonary Veins.* In most cases of thrombophlebitis, the lungs form a formidable barrier to the entrance of septic emboli into the arterial circulation.

A varying dosage of organisms not attached to larger particles of fibrin, etc., however, may pass through the lung capillaries and enter the left side of the heart. From there, they are distributed over the body by the arterial circulation and undoubtedly are being constantly removed by the kidneys, spleen and bone-marrow which are anatomical organs of elimination; and not, as Rosenow thinks, due to selective affinity of certain strains for such tissues.

Positive and negative blood cultures are, therefore, obtained throughout various stages of the disease and are of little prognostic value. If the dosage which passes through the lungs is small, and organisms are rapidly removed from the systemic circulation, negative blood cultures are apt to occur. The same happens when the thrombophlebitis in the original site of infection subsides, heals or is shut off from the circulation.

Provided there is no establishment of a secondary or tertiary focus of infection in the arterial system, such as an endocarditis, no embolic lesions will be encountered in the various viscera and body tissues.

There will be found, however, acute cloudy swelling in the heart muscle, kidneys and liver, an acute splenitis and often a severe anemia of the hemolytic type with attempts at bone-marrow regeneration.

### *Embolic, Metastatic Lesions*

*Acute Bacterial Endocarditis of the Mitral or Aortic Valves.* Most patients with thrombophlebitis die before a left-sided endocarditis is established.

The following embolic, metastatic lesions in the arterial circulation may, however, be encountered in some cases. They depend on the establishment of a secondary nidus in the endocardium.

In acute bacterial endocarditis, no matter what the invading organism may be,

the lesion produced is practically the same. The organisms lodge on the mitral, aortic or both valves with the same result. If the valves have been previously injured by rheumatism or congenital lesions they are more apt to be attacked.

The vegetations are as a rule large, soft, exuberant, more friable than in subacute forms of endocarditis and do not involve the mural endocardium. Final ulceration, perforation and destruction of the valve is common. The periphery of the vegetations is covered with agglutinated bacteria which are continually being discharged as emboli into the blood stream.

Acute bacterial endocarditis is always fatal and patients rarely survive six weeks after infection of the endocardium.

*Emboli to Myocardium.* There may be a direct extension of the infection on the valves to the adjacent myocardium, or emboli containing agglutinated organisms, etc., may break off and enter the coronary circulation, producing a shower of emboli throughout the heart muscle.

If the organism is the Streptococcus hemolyticus, septic emboli in the myocardium are rarely seen with the naked eye but sometimes can be recognized microscopically as small collections of leucocytes surrounding colonies of organisms.

If the organism is the staphylococcus, small, definite abscesses may be plainly seen with the naked eye. As this organism rapidly produces pus, the liquefied centers in subepicardial abscesses may project above the surface of the heart.

*Extension of Infection to Pericardium.* In a manner similar to the production of empyema from hematogenous abscesses of the lungs, these myocardial lesions may produce infection of the pericardium, by contiguity or by actual rupture. Usually a serofibrinous pericarditis is first noticed. It is a grave sign and of prognostic value. If, for instance, in a case of osteomyelitis a pericardial friction rub is heard, the outcome is almost always fatal.

If the patient lives long enough, a definite empyema of the pericardium may be found.

*Emboli to Nervous System.* From the aortic arch, emboli may gain entrance to the large vessels of the neck and be taken to the brain where septic infarcts may be seen both in the gray and white matter.

If the patient lives any length of time, providing this is not a terminal event, multiple brain abscesses usually develop. They are often small and their age can often be estimated by the thickness of the capsules.

Small petechial hemorrhages about the colonies of organisms in the central vessels occur. Sometimes, a large cerebral hemorrhage occurs, its origin being due to a septic infarction. It behaves clinically like an ordinary apoplexy.

A suppurative meningitis may occur, from infection of the meninges over such an abscess, by contiguity. Organisms may seep through the choroid plexus and drain down to the basal cysterna.

It should be recalled that in bronchiectasis, multiple brain abscesses occur in a similar manner from thrombosis of the small pulmonary veins. Large single brain abscesses are usually due to chronic ear infections.

*Splenic Infarcts.* The spleen is a common filter of emboli from the arterial circulation. In addition to the acute, swollen spleen, in these cases denoting the general sepsis and toxemia, infarction is common when the endocardium is involved.

The infarcted areas are usually soft and purulent and not the typical, firm, wedge-shaped yellowish infarcts seen in subacute bacterial endocarditis. A suppurative perisplenitis, in lieu of the adhesions seen over the infarcted areas in subacute endocarditis, is occasionally seen.

*Renal Infarcts.* Likewise, the kidneys are common filters of bacteria from the arterial system.

Renal septic infarcts may be similar to those seen in the spleen; they are purulent and the cortex of the kidneys may be riddled with small abscesses producing a condition of suppurative nephri-

tis. Masses of cocci may be seen lodging in the glomeruli.

In a manner somewhat similar to the production of empyemas in the pleural or pericardial cavities, the perinephric tissues may become infected from these septic emboli and a perinephric abscess of considerable size develop.

*Cutaneous and Mucous Membrane Emboli.* Embolic petechial hemorrhages may occur in the skin and mucous membranes. They should be looked for in the ocular and palpebral conjunctivae, on the sides of the neck, over the body and lower extremities and in the mucosa over the palate and cheeks. They may be innumerable or only occasional.

In streptococcal infections they appear as small bright red spots, 1–2 mm. in size, often with a slight fleck in their nonelevated centers.

In staphylococcal infections their centers are often raised, yellowish in color and surrounded by a red edge, due to rapid pus production by this organism.

In addition, small nodular swellings may occur on the soles and palms, often slightly bluish in color and not tender, in contradistinction to the Osler nodes of subacute bacterial endocarditis. These have been named by Libman[5] as Janeway lesions.

In some cases the petechial rash may be distinctly purpuric in character and, at times, render the diagnosis difficult.

At autopsy similar septic emboli may be seen in the serous cavities, especially over the pericardium, pleura, in the mediastinal tissues and peritoneum. They must be distinguished from asphyxial and purpuric lesions.

*Embolic Lesions in Eyes.* Petechial hemorrhages may be seen often in the retina by ophthalmoscopic examination when none can be found elsewhere in the body.

The uveal tract may be infected with the formation of a panophthalmitis.

*Embolic Lesions in Joints.* One or more joints may be the seat of a metastatic suppurative arthritis, the shoulder, knee and elbow being common locations. These joints may be distended with watery or thick pus without much pain, swelling or heat.

*Osteomyelitis.* The bone marrow, even in adult life, has a large capillary filtering bed in its red marrow, and, due to phagocytic cells of the reticulo-endothelial system, is also an important tissue in the purification of the arterial blood.

An embolic, pyogenic osteomyelitis can, therefore, occur in any bone, but usually in those which have during adult life considerable red marrow. The lesion is primary in the marrow in most cases, occasionally subperiosteal.

*Embolic Lesions in Arteries.* Finally, emboli may lodge in the peripheral arteries, especially those of the lower extremities, and produce, due to occlusion, gangrene. Rarely, septic emboli may lodge on the intima of arteries, usually the seat of a previous lesion of arteriosclerosis, producing a mycotic aneurysm with often the formation of a false aneurysmal sac filled with a septic clot.

### UTERINE ERYSIPELAS

A fulminating sepsis, sepsis foudroyant (Williams), acute lymphatic sepsis (Tweedy and Wrench), or what I have termed *uterine erysipelas* is a rare type of puerperal infection at the present time.

Before the days of aseptic obstetrical technique, in the days of midwifery, when the physician delivered women and treated cases of erysipelas and other dirty wounds on the same day, the condition was very frequent.

In 105 cases of suspected criminal abortion, this form of infection was not encountered. I have, however, seen it occasionally in puerperal infections.

The true disease is almost invariably fatal, death taking place in eighteen to thirty-six hours after delivery, the patient rarely surviving a week. Local lesions do not have time to form. Death seems to be due to a so-called toxemia, the meaning of

which, to me, has always seemed moot. Blood cultures by the ordinary technique are usually negative.

Clinical Course. The symptoms are similar to those of wound infections characterized by acute lymphatic sepsis, for example, postmortem infections which may kill in a few days.

The onset, after an incubation period of thirty-six hours or more, is usually characterized by a chill. The temperature rises and when once raised remains above normal. There is headache, the patient is thirsty, hot and flushed.

Characteristically the pulse, temperature and respirations all rise steadily, continuously and unabated as death approaches; although the patient is often apprehensive, euphoric and somewhat querulous, the psyche remains clear to the end.

The uterus involutes fairly well and is not tender. The lochia is much diminished and may be absent; it is not fetid. Diarrhea, abdominal distention and vomiting may all occur without peritonitis.

Autopsy Findings. The following pathological events are often seen:

*Infection of Endometrium with Streptococci.* All the cases are due to a hemolytic streptococcus infection. Although I have never bacteriologically proved that the infecting organism is the Streptococcus erysipelatis, I am convinced that such may be the case.

In 1929, I reported a case of phlegmonous gastritis as one of erysipelas of the stomach, after Birkhaug had definitely isolated the strain of streptococcus by means of absorption of antibodies and the production of a potent exotoxin which was completely neutralized by the antitoxin for S. erysipelatis.

The uterus, in contradistinction to other types of infection, is unusually clean, the endometrium being smooth and firm. Aside from the slight enlargement, it may appear almost normal to the naked eye. It usually, however, is enlarged, clean, brawny and edematous.

*Drainage into Lymphatics of Uterine Wall.* There is a bilateral, almost symmetrical involvement of the entire lymphatic system of the uterine walls producing a boggy, pale, edematous condition. This may only be appreciated when microscopic sections are studied, since, although the lymphatics are invaded by streptococci, the defense reaction of leucocytic infiltration is either scant or absent.

*Drainage into Lymphatics of Broad Ligament.* Likewise, the entire lymphatic system of both broad ligaments is filled with streptococci; however, there is no resultant peritonitis or tenderness.

*Drainage into Regional Lymphnodes of Pelvis and Pelvic Brim.* Extension rapidly takes place to the lymphnodes draining the uterus and the pelvis, especially those at the pelvic brim and around the bifurcation of the aorta which are enlarged, swollen, pale and edematous. Microscopic sections show them to be filled with streptococci.

*Drainage into Lumbar Lymphnodes and Thoracic Duct.* In cases which are extremely virulent, or in those who live longer than usual, an extension to the lumbar lymph nodes and receptaculum chyli may be seen.

*Drainage into Left Subclavian Vein.* In rare cases, after running a clinical course characteristic of this type of infection, the fever may break and become spiked in character. We must assume in such a case that the infected thoracic duct has eventually discharged a considerable dosage of streptococci into the subclavian vein and a septicemia has been superimposed.

I have seen recovery in one such case, in which the clinical diagnosis of this event was made, the previous negative blood cultures becoming positive. The woman recovered after several months of sepsis.

*Other Lesions Due to Toxemia.* The rest of the body shows lesions occurring in any severe toxemia. There is acute cloudy swelling in the heart muscle, kidneys and liver, together with an acute splenitis, but no emboli.

### PUTRID ENDOMETRITIS

In putrid endometritis, the infection usually remains localized in the uterus which is lined with a thick layer of necrotic material teeming with organisms. The cavity is filled with a profuse, foul-smelling bloody fluid containing gas bubbles.

Microscopic examination shows a very thick zone of leucocytic defense beneath this lining which usually limits the infection to the uterus.

In these cases, the invading organisms are putrefactive bacteria, colon bacilli, ordinary pyogenic cocci of low virulence, and gas-producing anaerobes, especially B. welchii.

If a careful bacteriological study could be made of these cases, as was done by Weinberg and others in gas gangrene during the world war, undoubtedly other gas-producing anaerobes could be isolated, particularly Vib. septique, B. oedematiens and B. sporogenes. This is a fertile field for research as, I believe, many of the rapidly necrotic and liquefying lesions in the uterine walls are due chiefly to anaerobic infection.

While all these organisms are introduced at one time, the pyogenic organisms and colon bacilli grow first and prepare the terrain for the rapid invasion by anaerobes. Each bacterium has an acidity of arrest peculiar to itself; for instance, when the tissues reach a certain acidity, streptococci stop growing and gas anaerobes which have been present, but dormant, rapidly multiply.

Many of these patients undoubtedly recover and run a clinical course known as *sapremia*. The more complete our bacteriological investigations are, the less significant sapremia becomes.

In fatal cases where the gas anaerobes become victorious, rapid liquefaction of the muscularis with extensive dissociation of tissues and gas cysts throughout uterine walls take place. Rapid extension to the peritoneum occurs and the profound toxemia often kills in twenty-four to thirty-six hours.

In some cases, an antemortem gas septicemia takes place; the skin during life becomes a mahogany color with marked distention of the abdomen. Autopsy, even when performed immediately after death, shows extensive destruction of the uterus, liver and spleen with gas cysts, these organs having the appearance of a red rubber sponge. The venous blood is filled with gas bubbles.

In 105 abortion cases, putrid endometritis with associated anaerobic infection was the cause of death in 8 cases (7.6 per cent). It is even more frequent in puerperal sepsis.

### TREATMENT

As the treatment of severe puerperal infections is almost hopeless, our greatest efforts should be directed toward its prevention.

It is not in the scope of this paper to discuss this most important phase further. The reader is referred to the recent report on maternal deaths in New York City.[6]

Kosmak[7] states "The high death rate associated with childbirth in the United States has been made the subject of very extensive comment, much of it being of a most uncomplimentary nature."

### SUMMARY

In this brief article, I have tried to lay stress on the fact that the problem of puerperal infection is almost entirely anatomical, depending on anatomical pathways for its spread, on physical and mechanical phenomena of stasis, drainage, etc., on cellular reactions called forth for defense, etc., and has very little to do with fancy theories of immunity, selective affinity of certain strains of bacteria for certain tissues, etc.

In addition, the condition of septicemia, which denotes only the ability to recognize bacteria in the circulating blood, is in many of these cases intermittent, and, at times, does not exist. I have endeavored to enumerate the anatomical reasons for this assumption.

In other words, bacteria do not multiply and grow in the blood but are discharged into it from either primary, secondary or tertiary depots in the various organs, in usually an intermittent fashion.

When thrown into the circulating blood, nature makes every effort to quickly rid the blood of their presence through its organs of elimination and filtration. The application of chemotherapy in the intravenous injections of mercurochrome, gentian-violet, and other like substances, is only excusable on the premise of attempting to kill those organisms circulating in the blood at the time of the injections, and to thus prevent the establishment of secondary foci in other parts of the body. As one is unable to know when the blood is being showered, the hopelessness of such procedures seems convincing.

Medication is, then, of no avail; vaccines and sera are equally useless. Only supportive blood transfusions seem to be of any benefit. I am not competent to discuss the value of artificial fever or heat produced by physical means.

Williams[2] states, "In such cases prayer is the best treatment, and the better prayer a doctor is, the better his infected patients do." The more radical the treatment the higher the mortality. The best results are obtained by those who do the least.

While the anatomical view of the subject leads one immediately to the possibility of radical surgery in its irradication, the obvious difficulty in knowing how far the process has extended greatly restricts surgery to the incision and drainage of localized collections of pus (for instance, an abscess pointing above Poupart's ligament), and in rare, selected cases where either the immediate ligation of thrombosed veins or a quick hysterectomy for a uterus containing multiple abscesses, might be seriously considered. According to Williams the latter two surgical procedures were in his experience indicated only once in every few years.

Finally, although this paper has been somewhat limited to the infections of the female pelvis, the general anatomical considerations, in the main, are applicable to other pyogenic infections, more especially to those of the middle ear and accessory sinuses of the head and to infected wounds of the extremities and trunk; the difference lying only in the type of tissues affected.

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In preparing this sketch I have tried mainly to give my experience as a pathologist and medical examiner, disregarding almost entirely the extensive literature.

In addition to the references noted above, I have freely used opinions and arguments from the following authors, to whom I wish to acknowledge my indebtedness:

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# EPIDEMIC OF FATAL ESTIVO-AUTUMNAL MALARIA

## AMONG DRUG ADDICTS IN NEW YORK CITY TRANSMITTED BY COMMON USE OF HYPODERMIC SYRINGE*

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THE outbreak of an epidemic of fatal estivo-autumnal malaria confined entirely to the drug addict population in Manhattan, and the peculiar mode of transmission of the disease in a locality where anopheles mosquitoes are almost non-existent and during a season of the year when malaria ordinarily does not occur, has prompted the writing of this report.

During a five months period, from September 25, 1933 to February 28, 1934, 20 fatal cases of estivo-autumnal malaria of the cerebral type, and one fatal case of quartan malaria complicated by bronchopneumonia were autopsied by the Office of the Chief Medical Examiner. An additional autopsied fatal case and a non-fatal case of the cerebral form of estivo-autumnal malaria occurred at the United States Marine Hospital at Ellis Island.[1] During the same period, 18 non-fatal cases of estivo-autumnal malaria and 8 non-fatal cases of quartan malaria, a type very unusual in this part of the world, and one case of tertian malaria were also discovered. In all, there were 49 cases, 39 of which were estivo-autumnal. The distribution of the cases is indicated in Table 1.[2] In every instance the disease occurred in a drug addict who injected heroin intravenously, the so-called "main line shooter." In a brief communication[3] to the Surgeon General on February 8, 1934, 41 of these cases were reported.

A digest of the clinical and pathological findings in the 21 fatal estivo-autumnal cases and the results of an epidemiological study of all the malaria cases will be presented in this paper. The clinical details of the 9 quartan cases will not be included. During the course of preparation of the 49 cases in the present series, the clinical histories of the 4 quartan and 6 of the 7 estivo-autumnal cases which occurred on the Fourth Medical Division of Bellevue Hospital were recently reported by Applebaum and Gelfand.[4] They did not mention that the 6 fatal estivo-autumnal cases in their group were autopsied by the Office of the Chief Medical Examiner.

The first case which came to our attention will be briefly narrated.

On October 9, 1933, A. K., a white man aged thirty, a seaman residing in a hotel on South Street, was admitted to the Second Medical Division of Bellevue Hospital. He had been found lying semi-comatose on a park bench. A diagnosis of drug poisoning was made by the ambulance surgeon who noted hypodermic needle scars over the veins of the left antecubital fossa. No history was obtainable. On admission, the patient was semi-comatose, unable to respond to questioning and exhibited a withdrawal response to painful stimuli. The face was flushed, the lips dry, the respirations deep and slow, the pulse full and 88 per minute. The temperature was 97°F. The blood pressure was 110 mm. Hg systolic and 60 mm. diastolic. The eyeballs were slightly softer than normal, the pupils were dilated and reacted to light, the jaws were clenched. The heart was regular and the lungs clear. The abdomen was soft, the liver and spleen not palpable. The extremities were thin and the deep reflexes active. The urine contained one plus sugar and 3 plus acetone. The blood sugar was 295 mg. per cent. The spinal fluid was clear but under increased pressure; 12 lymphocytes were found per cu.

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mm.; globulin was absent and a normal amount of reducing body was present.

Because of the blood sugar and urinary findings, a diagnosis of diabetic coma was made. Glucose and insulin were administered following which the urine became sugar and acetone free. The next day, the right eye turned downward and inward, the extremities were hypertonic, the deep reflexes were slightly increased and the abdominals were absent. The discs in both fundi were hazy. Stimulants and fluids were administered but the patient became progressively weaker, the pulse rapid and thready and the respirations shallow. The temperature rose from 97°F. to 101°F. and then rapidly to 108°F. Death occurred thirty-six hours after admission. The final diagnosis was unknown poisoning, possible brain injury or tumor involving the fourth ventricle. Because of the evidence of drug addiction, this and all the other fatal cases were referred to the Office of the Chief Medical Examiner for investigation.

At autopsy, extensive tattooing of the names of many tropical ports was noted and drug addiction was evident from the numerous pigmented needle scars over the veins of the left forearm. The skin was sallow, the body poorly nourished. The spleen was moderately enlarged, weighing 350 gm., very soft, the pulp mushy and dark reddish brown in color. The liver weighed 1750 gm. and was bluish brown in color. The lungs were edematous and congested. The surface of the brain was reddish gray brown in color. There was intense pial congestion. The brain substance was soft and swollen and on section numerous punctate hemorrhages were distributed throughout the white matter of the cerebrum, in the internal capsules and throughout the cerebellum. The cortex was congested and darker than normal. On microscopic examination, the capillaries everywhere in the brain were dilated and filled with red blood cells containing pigmented malarial parasites in great number. Clumps of malarial pigment grains were phagocytosed in swollen endothelial cells. There were small hemorrhages around capillaries plugged with parasitized red cells. In the white matter, small glial cell proliferations, the so-called malarial granulomata, were found around some of the vessels. Small areas of demyelinization and slight swelling of the nerve cells were noted. In the liver, the Kupfer cells were swollen and contained numerous phagocytosed malarial pigment grains. In the spleen, phagocytosis of pigment by the pulp cells was also seen. A diagnosis of estivo-autumnal malaria with cerebral localization of parasites was made.

## EPIDEMIOLOGY

When this case was recognized at autopsy the possibility of a relapse of a naturally acquired tropical malarial infection in a seaman who had been to the Tropics was strongly considered. However, there occurred a rapid succession of fatal cases with almost identical histories and pathological findings, always in drug addicts showing evidence of administration of drugs by the intravenous route. These findings indicated that the association of drug addiction and the same type of malaria was more than a coincidence. A prediction that more cases would soon appear and that in all instances the patients would be drug addicts, was quickly borne out. At the same time, investigations carried on with the assistance of Detective Jocker of the Narcotic Squad and Detective Oswald of the Homicide Squad of the Police Department revealed that several of the fatal cases had never been away from the vicinity of New York City and that many of the fatal cases, including the one from Ellis Island, were known to each other and had frequented the same lodging houses; also that several of the deceased had been to the Tropics.

These facts pointed to a direct transmission of the disease from individual to individual and a knowledge of the technique of intravenous injection of drugs employed by addicts readily explained how it occurred. The syringe or "works" (Fig. 1) is usually improvised by inserting a medicine or "eye" dropper into a hypodermic needle which is designated as a "spike." An air tight fit is obtained by wrapping a small piece of newspaper around the tip of the dropper before inserting it into the needle. This apparatus is frequently used in rapid succession, without preliminary washing or sterilization,

by two or more addicts for intravenous injection of a solution of heroin. Frequently several addicts, not having sufficient money

Fig. 1. "The works." Syringe for intravenous heroin injection improvised from medicine dropper and hypodermic needle. Solution is prepared in bottle cap and then drawn up through wad of cotton which serves as a filter. Apparatus is carried in tobacco bag.

to buy the drug individually, pool their funds to buy a single portion or "shot" of drug which is then dissolved in water and drawn up into the syringe, each addict injecting his share of the solution. When the needle enters the lumen of the vein a quantity of blood always flows back into the syringe. In this manner, a malarious addict introduces blood containing malarial parasites into the syringe. The addict who next uses the apparatus injects some of this blood into his vein and in this very simple manner inoculates himself with whatever type of malaria his associates may have. He in turn becomes a carrier capable of transmitting the disease in the

Fig. 2. Pigmented needle puncture scars along course of sclerosed superficial veins of antecubital fossae and forearms. Intravenous drug addict. "Main line shooter."

same manner in which he acquired it. As pointed out by Himmelsbach,[5] although it is essential that the "donor" must withdraw blood for transmission of the disease, it is possible for the "recipient" to infect himself also by subcutaneous injection.

The method of taking the drug intravenously is comparatively new in New York City but has been practiced for many years in other localities. The direct transmission of 10 cases of malignant malaria among drug addicts using the intravenous route for injection of drugs was first described in 1929 by Biggam[6] in Cairo, Egypt. In 1930 Biggam and Arafa[7] reported more than 100 cases of malaria transmitted in this fashion. In this country, Geiger[8] in 1932 was the first to call attention to the probable direct transmission of malaria among drug addicts sharing the use of syringes after the appearance of 5 cases of malaria in addicts in the San Francisco Hospital. In 1933 Nickum[9] in Omaha, Faget[10] in New Orleans, Flaxman[11] and Eaton and Feinberg[12] in Chicago, and

TABLE 1
CASES OF MALARIA IN DRUG ADDICTS IN THE BOROUGH OF MANHATTAN, CITY OF NEW YORK
September 25, 1933 to February 28, 1934

| *Type* | *Place* | *Fatal Cases* | *Non-fatal Cases* | *Total* |
|---|---|---|---|---|
| *Estivo-autumnal* | Bellevue Hospital—First, Second, Third and Fourth Medical Divisions and Psychopathic Hospital | 16 | 8 | 24 |
| | U.S. Marine Hospital, Ellis Island | 1 | 1 | 2 |
| | Correction Hospital, Welfare Island | 2 | 6 | 8 |
| | Gouverneur Hospital | 1 | 1 | 2 |
| | Lodging House | 1 | 0 | 1 |
| | Private Physician | 0 | 2 | 2 |
| | Total | 21 | 18 | 39 |
| *Tertian* | Correction Hospital, Welfare Island | 0 | 1 | 1 |
| | Total | 0 | 1 | 1 |
| *Quartan* | Bellevue Hospital—Second and Fourth Medical Divisions and Psychopathic Hospital | 1 | 3 | 4 |
| | Correction Hospital, Welfare Island | 0 | 5 | 5 |
| | Total | 1 | 8 | 9 |
| | Totals | 22 | 27 | 49 |

Himmelsbach[5] at Fort Leavenworth reported cases of direct transmission of malaria among drug addicts. Faget also mentioned the occurrence of several cases in Galveston. In his New Orleans series there were 5 fatalities, all probably estivo-autumnal, and 5 non-fatal cases, 4 of which were estivo-autumnal and 1 a quartan.

The accidental direct transmission of estivo-autumnal malaria among a group of 10 patients receiving salvarsan injections from a common gravity injection apparatus in which the needles but not the glass window in the tubing were changed, was described by Wenyon[13] in 1926. Instances of accidental transmission of malaria during blood transfusion have been recorded by Korabelnikoff,[14] Decourt,[15] Netter[16] and others. It is of interest that the first report of the successful direct inoculation of malaria by injection of malarious blood was made by Gerhardt[17] in 1884.

Following the appearance of the first fatal cases of estivo-autumnal malaria in drug addicts, it became reasonably certain that the disease was directly transmitted within that group, as a result of the peculiar practice of sharing unsterilized syringes for the intravenous injection of drugs. The existence of a number of latent cases was anticipated. Because of this, a malarial survey of a group of drug addicts at the Tombs Prison and at Correction Hospital was carried out on November 28, 1933, with the permission of former Deputy Commissioner of Correction Tudor and with the assistance of Dr. Barland, the resident physician at Correction Hospital. In a routine examination of blood smears from 150 addicts not suspected of having malaria, 9 individuals were found who harbored malarial parasites in their blood; 8 of the 9 were estivo-autumnal and 1 was a tertian. On being questioned these carriers readily admitted having shared their syringes with each other and also with many of the addicts who had died of malaria. Several of these

TABLE II

DEFINITELY ESTABLISHED CONTACTS THROUGH COMMON USE OF SYRINGES AMONG DRUG ADDICTS SUFFERING FROM ESTIVO-AUTUMNAL MALARIA

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | A | B | C | D | E | F | G | H | I | J | K | L | M | N | O | P | Q | R | S | T | U |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | | + | + | | + | | | | | | | | | | | | | | | | | | | | + | | + | | | | | | | | | | | | | |
| 2 | + | | + | | | | | | | | | | | | | | | | | | | | | | + | | + | | | | | | | | | | | | | |
| 3 | + | + | | + | + | | | | | | | | | | | | | | | | | | | | | | + | | | | | | | | | | | | | |
| 4 | | | + | | + | | | | | | | | | | | | | | | | | | | | | | + | | | | | | | | | | | | | |
| 5 | + | | + | + | | | | | | | | | | | | | | | | | | | | | | | + | + | | | | | | | | | | | | |
| 6 | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | + | | | | | | | | |
| 7 | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| 8 | | | | | | | | | | | | | | | | | | | | | | | | | | + | | | | | | | | | | | | | | |
| 9 | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| 10 | | | + | | + | | | | | | | | | | | | | | | | | | | | | | + | | | | | | | | | | | | | |
| 11 | | | | | | | | | | | | + | | | | | | | | | | | | | | | | | + | | | | + | + | | | | | | |
| 12 | | | | | | | | | | | + | | | | | | | | | | | | | | | | | | + | | | | + | + | | | | | | |
| 13 | | | | | | | | + | | | | | | | | | | | | | | | | | | + | | | | | | | | | | | | | | |
| 14 | | | | | + | | | | | | | | | | | | | | | | | | | | | | + | | | | + | + | | | + | | | | | |
| 15 | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | + | |
| 16 | | | | | | | | | | | | | | | | | + | | | | | | | | | | | | | | | | | | | | | | | |
| 17 | | | | | | | | | | | | | | | | + | | | | | | | | | | | | | | | | | | | | | | | | |
| 18 | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |

Numerals 1 to 18 indicate non-fatal cases from whom information was obtained concerning common use of syringes for intravenous injection of drugs.

Letters A to U indicate the 21 fatal cases concerning whom information was obtained from the non-fatal cases. The common use of the syringe between any two addicts is indicated by the symbol +.

addicts stated they had never been out of New York City. One admitted recent malarial infection in the Tropics. On examination they were febrile, asthenic, and anemic, the hemoglobin readings varying between 40 and 60 per cent. All had chilly sensations, some had frank chills; 4 had palpable spleens; 6 were drowsy. They attributed their symptoms either to the effects of drug withdrawal or to adulterated samples of drug. Under quinine therapy, they all made an uneventful recovery.

From information obtained from the non-fatal cases and also from police witnesses, a chart (Table II) was constructed on which definite instances of contact between malarious addicts, through the common use of unsterilized syringes, are indicated. The symbol + represents an admitted instance of two addicts injecting drugs from the same unwashed syringe, or "shooting up together." In two instances the procedure was witnessed by a detective of the Narcotic Squad who then confiscated the

syringe and arrested the addicts. One of these addicts died in malarial coma shortly after arrest. A confiscated injection outfit is illustrated in Figure 1. An attempt to stain and examine the inner surface of the syringe for parasites was unsuccessful. This was also attempted by Biggam[6] in 1929 but without success.

Numerous contacts admitted by malarious addicts could not be indicated, the addicts in question not remembering or not knowing the person who shared the syringe. All admitted the practice. They stated that syringes were frequently placed in lavatories and other hiding places and used in rapid succession by several addicts who were not known to each other. The practice of hiding the syringe is a common one, the addicts thus avoiding arrest for possession of a hypodermic syringe. In some sections of the City, the syringes are kept in vacant apartments and the addict pays a small rental fee to the owner, who is also generally a drug peddler, for the use of the apparatus in privacy. Sterilization is never carried out. It is thus easy to understand how direct malarial infection can occur and the victim not know from whom he received his infection.

It was not possible to link the cases in this series with any of those that occurred in other epidemics elsewhere. One non-fatal case admitted having been in New Orleans in the Winter of 1932 when several cases of estivo-autumnal malaria among drug addicts were reported by Faget.[10]

Because of frequent contacts, no accurate estimate of the incubation period could be formed in any of these cases. According to Craig[18] the incubation period of estivo-autumnal malaria when artificially induced by intravenous injection is five to six days. Bignami[19] found it to vary from six to ten days and Bastianelli and Bignami[19] from three to five days. Thus it seems that there is considerable doubt as to the accuracy of the incubation period of two days given by Applebaum and Gelfand[4] in one of the cases which they report.

## CLINICAL DIGEST

The entire group of 49 addicts in this malaria epidemic were of the male sex and the majority were in the third and fourth decades of life. Of the 39 estivo-autumnal cases, 28 were white, 3 were black and 8 were Puerto Rican. At least 24 of the 39 showed evidence of homosexuality, a perversion frequently associated with drug addiction. Thirteen gave a lodging house on Park Row, 6 gave one on South Street, and 5 gave one on Sixth Avenue, as their addresses. The others were scattered in various inexpensive hotels in the lower part of Manhattan. By occupation, they were seamen, clerks, laborers, bus boys, soda clerks, orderlies, entertainers, electricians.

The majority of the patients were brought to the hospitals in ambulances. Of the 21 fatal cases, one patient died in coma in a lodging house after having refused hospitalization, and 2 were conscious when admitted, becoming stuporous shortly after. Another case was conscious on admission but died of a lobar pneumonia and empyema which developed several days after the malarial parasites disappeared from his blood. The remaining 17 fatal cases and 4 of the 18 non-fatal cases were admitted to the hospital in a semi-stuporous, stuporous or comatose condition. One such patient was maniacal and was taken directly to the psychopathic ward.

Of the 3 fatal cases that were conscious on admission, 2 had been transferred from one hospital to another, one having been sent for drug withdrawal treatment and the other with a diagnosis of grippe. Estivo-autumnal malaria was not diagnosed in either of these cases until after coma had set in.

Of the cases that were admitted in stupor or in coma, 4 were brought from lodging houses, 3 were picked up on the street, 1 was picked up in a subway station, 1 on a park bench, 1 was found crying in a moving picture theatre, 1 was brought from a restaurant where he collapsed while drink-

ing coffee, 2 were found in doorways, 2 became unconscious in the Tombs Prison, and 2 became stuporous shortly after arriving at the Penitentiary. As nearly as could be determined, the interval of stupor before admission to the hospital varied from several hours to three days.

The majority of the non-fatal cases on admission to the hospital were tremulous, febrile, complaining of chilliness, chills, headache, cough, generalized aches and pains. Many of these patients attributed their symptoms to the effect of drug withdrawal or to the use of an impure sample of drug.

*Physical Findings.* All of the fatal cases were acutely ill when first seen. Nutrition was poor. All but three were stuporous or comatose and did not respond to questioning. The eyes were generally open and the faces usually exhibited a staring expression. In several instances there was a fine purpuric eruption over the shins. Jaundice was noted only occasionally.

In all but 3 patients, evidence of intravenous drug addiction was noted in the form of closely placed punctate pigmented needle scars over the superficial veins of the arms, antecubital fossae and forearms (Fig. 2). In 2 of the 3 fatal cases in whom drug addiction was not suspected clinically, needle scars over the veins were apparent at autopsy. In the third case information that the deceased had been an addict was obtained from the police.

Eye Signs. Involuntary rolling of the eyeballs and lateral nystagmus were occasionally noted. The pupils were usually dilated and reacted poorly to light. In one case the pupils were small; at times they were slightly irregular. The fundi in most cases were reported as hyperemic, especially the discs which were reddish in color. The disc margins were occasionally hazy and at times exhibited very slight edema; the veins were engorged. In some instances the fundi were reported as normal. In 4 of the fatal cases, small flame-shaped retinal hemorrhages were seen. The fundi were not reported in 6 cases.

Neurological Findings. In almost every case, the deep reflexes were hyperactive. In one case with frequent Jacksonian epileptic seizures, the deep reflexes were abolished. In another instance they disappeared shortly before death. Clonic convulsions were noted twice just before death. Ankle jerks were absent in one case. The Babinski reflex was noted in 6 cases; ankle clonus in 5; the Kernig sign in 5; the Brudzinski sign in 3 cases. The abdominal reflexes were frequently diminished or absent. A variable amount of neck rigidity was noted in 10 instances; coarse tremors of hands and leg muscles in 4 cases. A generalized hyperesthesia to slight stimuli and a protective response to painful stimuli were observed in 12 cases. Muscle spasticity was noted in 7 cases and incontinence of urine and feces in 6 cases. Vomiting occurred in 2 cases; severe sweating in several instances.

The spleen was palpable in only 9 out of 17 of the fatal cases.

The blood pressures in the fatal cases were fairly low, most of the cases having a systolic pressure between 100 and 110 mm. Hg and a diastolic pressure between 60 and 80 mm.

Temperature. All of the fatal and non-fatal cases were febrile. In the fatal cases the temperature curves were extremely variable. In the main, 4 types of curves were noted; rapidly rising temperatures, falling temperatures, level temperatures, and fluctuating temperatures. Irregularly fluctuating temperatures were noted in 7 instances and in 1 case there was a daily swing of 5°F. from 99.4 to 104.2 with a gradual elevation of the upper and lower levels. Quinine therapy did not influence this temperature curve. In several instances there was a progressive rise from an initial normal or slightly elevated level varying between 97° and 102° to a height of 108° and 108.6°F., occurring within twenty-four to thirty-six hours. In several cases, an initial elevated temperature fell slightly before death, e.g., 103.6° to 100.6°, 103.6° to 101.6°; 102° to 101°F. A number

of temperature curves remained practically constant showing no fluctuation at all, e.g., 101.8°; 102.6° to 103.4°; 104.6° to 104°; 105.4° to 104.4°F.

The pulse rate was usually in proportion to the temperature, at times lagging slightly behind but usually exceeding the temperature curve at the end. The respirations were generally increased varying between 24 and 48 per minute. At times the breathing was labored, at other times irregular and shallow.

The duration of the illness in the fatal cases after hospitalization lasted anywhere from ten hours to four days; 5 patients died within twenty-four hours, 2 lived thirty-six hours, 6 lived forty-eight hours, 2 lived three days, 2 lived four days and 1 lived sixteen days dying of a lobar pneumonia and empyema which set in several days after the malarial parasites had disappeared from the blood and the temperature had dropped to normal. The latter case was treated with atabrine and was not comatose.

*Laboratory Findings.* Spinal fluid was examined in 15 of the fatal cases. In all but one, the fluid was clear and colorless; in one instance a bloody fluid was obtained. The pressure was noted as increased in 3 instances. The cell counts varied from 0 to 60 cells, all lymphocytes. A slight increase in globulin was reported in 2 cases; a 2 plus albumin was reported in 2 cases; reducing substance was increased in 5 cases. The Wassermann test made on 6 fluids was negative in 5 and slightly positive in 1. The colloidal gold reactions were done on 4 fluids and were normal.

Urinalysis. The urines recorded in 14 cases were all clear, amber and acid, the specific gravity varying between 1.018 and 1.032. Six urines contained 1 plus sugar; 3 urines contained 3 plus sugar after glucose infusion; 4 urines contained acetone; 1 tested urine contained urobilin in dilution 1 to 320. Microscopic examinations revealed occasional white blood cells in 6 urines and occasional red cells in 3 urines. Occasional hyaline and granular casts were found in 3 urines.

Blood Examinations. Erythrocyte counts were made in 10 of the fatal cases. The counts varied between 2,820,000 and 5,000,000. The hemoglobin determinations varied between 45 and 90 per cent; the color index was usually less than 1. The leucocyte counts in 13 cases varied considerably between 5200 and 46,000. Four counts were under 8000; 5 counts were between 10,000 and 12,000; 4 counts were between 15,000 and 19,000; 1 count of 46,000 were recorded. The differential counts were also variable. Immature granulocytes were noted in several instances. In the majority of instances, high leucocyte counts were associated with the presence of lobular pneumonia. The blood Wassermann recorded in 6 cases was positive in 2.

Blood smears were examined for malarial parasites in only 13 of the fatal cases, the disease not being suspected in 8. These smears revealed the presence of Plasmodia falciparum as young non-pigmented schizonts (ring forms) and also as crescentic gametocytes (Fig. 3). Crescents were not found in 2 cases. The infection of the red cells was variable, at times moderate, at times heavy. The overwhelming infections such as Seyfarth[25] describes in the septicemic forms of the disease were not seen. In 2 instances, development of the schizonts up to the merocyte stage was observed in the peripheral blood, an indication of a severe infection and seen occasionally in moribund cases. In all the other cases, only the small delicate non-pigmented young ring forms were found in the smears of circulating blood, the older pigmented schizonts having disappeared into the capillaries of the internal organs where segmentation of Plasmodium falciparum usually takes place. The ring forms were frequently eccentric or marginal and sometimes double in the red blood cells. In several smears, mononuclear cells containing phagocytosed malarial pigment grains, the so-called melaniferous leucocytes, were seen. The parasites were

usually easily found when looked for in most instances, where the disease was suspected. In several instances however, in the non-fatal cases where the disease was suspected, they were not found on the first smears but were discovered subsequently.

Blood Chemistry. In 13 cases, the non-protein nitrogen of the blood varied between 30 and 175 mg. per cent. In 11 cases the values were under 50 mg. The blood sugars varied between 60 and 295 mg. per cent; most of the readings were normal. The significance of the isolated blood chemistry findings is doubtful in view of the amount of dehydration present in most of the patients and in the absence of significant findings in the urine.

*Diagnoses.* It is easily understood how the aforementioned clinical signs and symptoms in the absence of a blood smear examination for malaria would give rise to the following diagnoses which were made in the first 7 undiagnosed cases: drug withdrawal symptoms; drug poisoning; toxic encephalitis; septicemia following drug injection; pneumonia; typhoid or paratyphoid; diabetic coma; psychosis with organic brain disease; meningococcic septicemia; acute encephalitis; tuberculous meningitis; cerebral thrombosis; hemorrhage into a glioma; brain abscess; miliary tuberculosis; grippe; influenza; epilepsy.

When, as a result of the autopsy findings, attention was called to the prevalence of the disease, subsequent cases in almost every instance were promptly diagnosed and confirmed by the presence of Plasmodia falciparum in the blood smears.

*Treatment.* Of 16 recognized cases admitted to the hospitals in stupor or in coma, only 4 responded to intravenous quinine therapy. One of these received both quinine and atabrine.[4] The remainder of these comatose cases did not respond even to intensive intravenous quinine therapy, or, in two instances to the use of atabrine.[4] In all cases diagnosed before coma developed, ordinary quinine therapy was successful and the patients promptly recovered. In one such patient who was treated with atabrine, the temperature became normal and the plasmodia disappeared

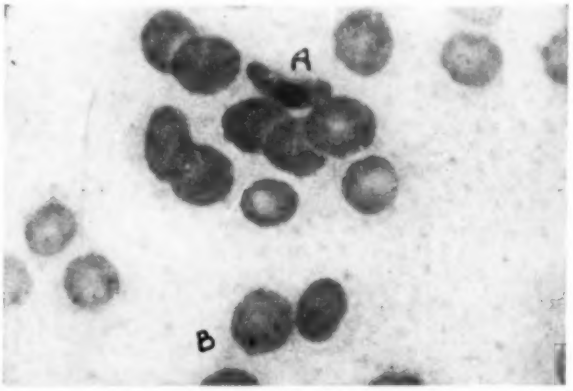


Fig. 3. Blood smear from case of estivo-autumnal malaria in series. Crescentic gametocyte at A and small non-pigmented ring forms in a red cell at B. Giemsa stain. Oil immersion.

from the blood smears, but the patient subsequently died of a lobar pneumonia and a massive serofibrinous pleuritis.

## PATHOLOGICAL DIGEST

The findings in the 21 autopsied estivo-autumnal cases were fairly constant. The bodies were poorly nourished, the tissues dehydrated and the skin and mucous membranes pale and sallow. Tattooing was frequent. In every case, slightly pigmented needle puncture scars were present over the veins of the antecubital fossae and forearms. Occasionally cutaneous needle scars were found on the arms and thighs.

The brains, examined in all but one case, were heavy and soft. The dura was tense, and the cerebral convolutions moderately flattened. The pia arachnoid was transparent and moist and the pial blood vessels intensely injected. The color of the brains varied but generally the cortex presented a striking reddish gray brown appearance. In some instances the color was almost orange, in others, dusky gray brown. When sectioned, the cortex was strikingly demarcated from the white matter. In many of the brains (Fig. 4) innumerable closely placed punctate hemorrhages were found in the cerebral white matter, especially

in the subcortical regions. When numerous, these hemorrhages were also seen in the internal and external capsules and were

Fig. 4. Horizontal section through brain of drug addict dying of estivo-autumnal cerebral malaria. Cortex is darker than normal and numerous punctate hemorrhages are scattered throughout white matter.

sprinkled throughout the basal ganglia and mid-brain. In the cerebellum, they were found just beneath the pia, in the cortex, in the granular layer, in the white matter and in the dentate nuclei. In some of the brains, the punctate hemorrhages were scanty in the cerebral white matter and readily made out in the cerebellum. In a few cases, no hemorrhages were found. No gross hemorrhages or other changes in the brain substance were noted. In one instance, a thin subarachnoid hemorrhage was found over the left temporal lobe. The lateral ventricles contained clear fluid.

In every instance, where crushed smears stained with Giemsa were made from any part of the brain substance, the capillaries were found to contain large numbers of pigmented Plasmodia falciparum within red cells. These plasmodia exhibited all stages of asexual development including non-pigmented merozoites, older pigmented schizonts and segmenting merocytes or rosettes (Fig. 5). The endothelial cells of the capillaries were swollen and intracapillary phagocytes containing

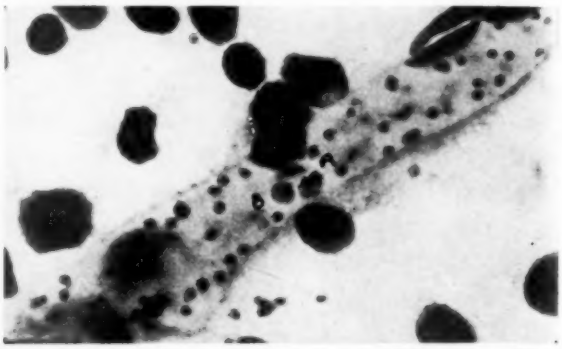

Fig. 5. Smear of crushed brain tissue. Dilated capillary containing red blood cells infected with plasmodia. A merocyte or rosette at A, a pigmented schizont at B. Giemsa stain. Oil immersion.

parasites and pigment clumps were found. Parasites were not found in the brain of the non-comatose case that died of lobar pneumonia, nor in the fatal quartan case.

In the microscopic sections of brain tissue a striking feature was the extreme dilatation of all the capillaries. These dilated vessels were laden with red cells containing enormous numbers of pigmented plasmodia (Fig. 6). In the larger capillaries and in the arterioles, a peripheral distribution of all the infected red cells was found. Many endothelial cells were swollen and contained clumps of phagocytosed pigment. In places, the capillaries were occluded by agglutinated parasitized red cells or by agglutinated red cell thrombi in which parasites were not numerous. The punctate hemorrhages noted grossly were composed of non-infected red cells although they surrounded capillaries filled with infected red cells. In the larger of these capillary hemorrhages, parasitized red cells, malarial pigment grains, blood pigment and pigmented macrophages were sometimes visible. In the cerebral and cerebellar white matter, focal proliferations of glial cells, the malarial granulomata described by Dürck[20] were found surrounding and adjacent to capillary vessels. In places the glial cells were arranged in

long single rows along the sides of the capillaries. Small areas of demyelinization were seen. The nerve cells exhibited degenerative changes which were not severe. The pia arachnoid was congested and edematous and infiltrated with mononuclear cells. The small pial blood vessels contained numerous infected red cells and pigmented macrophages.

The spleens were enlarged, blue gray or slate colored, soft and swollen. The weights varied between 240 gm. and 650 gm. The pulp was dark reddish brown, very mushy, and the follicles were indistinct. In stained smears, an increase in pulp cells, free and phagocytosed pigment grains, red cells containing developing schizonts and young and mature gametocytes were seen. The infection of the red cells was not heavy. In microscopic sections, an increase in pulp cells and abundant fairly evenly distributed free and phagocytosed malarial pigment grains were found in the pulp spaces. Pigmentation was not noted in the splenic follicles.

The livers were enlarged, softer than normal, slate colored, varying in weight between 1550 and 2350 gm. The microscopic sections revealed considerable swelling of all the Kupfer cells, the cytoplasm of which was laden with phagocytosed malarial pigment grains.

The bone marrow at the lower end of the femurs did not show any obvious brown pigmentation such as is found in chronic malarial infections. The rib and sternal bone marrow were dark red in color and slightly mushy. Smears of the femoral marrow revealed only occasional parasites and pigment grains and sections revealed parasitized red cells and phagocytosed pigment in the marrow capillaries, but not in great amount. Smears and sections of the rib and sternal marrow were much more cellular but parasites and pigment deposits were not abundant. Young ring forms and older pigmented schizonts and young and old crescents, and occasional pigmented macrophages were seen in the smears.

The lungs were congested, heavy and edematous. Hypostatic lobular pneumonia, purulent bronchitis and bronchiolitis were found in many instances. As already mentioned in one case a massive lobar pneumonia and serofibrinous pleuritis were found.

Fig. 6. Section of cerebral cortex. Dilated capillary vessels containing red blood cells heavily infected with pigmented plasmodia. Hematoxylin and eosin stain. High dry.

The hearts were generally small or normal in size, usually dilated, and on microscopic examination were not unusual.

The kidneys were slightly swollen, reddish brown in color and on microscopic section, phagocytic cells containing pigment grains were found scattered throughout the glomerular tufts. A moderate amount of granular degeneration of the tubular epithelium was noted.

No excessive localization of malarial parasites was found in the capillaries of the heart, lungs, kidneys, pancreas, adrenals, thyroid, lymph nodes, testes and gastrointestinal tract.

The pathological diagnosis in the above cases was estivo-autumnal malaria with cerebral localization.

## COMMENT

The clinical features and pathological findings of the cerebral form of estivo-autumnal malaria have been completely described by many observers[20—25] in the past. The observations recorded in the present paper merely confirm their findings.

Since Ewing[22] reported his investigations of many fatal cases of naturally acquired estivo-autumnal malaria, which occurred chiefly in soldiers returning from Cuba after the Spanish American War, there has been no significantly large number of fatal cases of the disease in this locality. The rarity of estivo-autumnal malaria in this climate and its protean clinical manifestations which simulate many other diseases, were responsible for the incorrect diagnoses of 7 of the first cases to appear at Bellevue Hospital.

The cerebral involvement, the extremely soft and only moderately enlarged spleens and the lack of pigmentation in the bone marrow were findings consistent with the fulminating acute course of the disease in the fatal cases. The coma was produced in every instance by the localization of the parasites in the brain capillaries. The high mortality of the cerebral type of estivo-autumnal malaria after coma has set in, even with intensive anti-malarial therapy, is in agreement with the findings of others,[22,23,24] experienced in the treatment of this dangerous type of the disease.

It is interesting and perhaps significant that all 20 fatal cases of estivo-autumnal malarial should have died in coma as a result of cerebral involvement and not have shown any of the manifestations of the other well defined clinical types of the disease. Seyfarth[25] has divided deaths from estivo-autumnal malaria into several groups depending upon the distribution of the parasites in the body. He states that about 30 per cent of all fatalities result from overwhelming blood stream infection, the so-called septicemic type; about 55 per cent of all fatalities are of the cerebral type with massing of parasites in the capillaries of the brain; about 14 per cent are of the cardiac type with localization in the capillaries of the heart; about 1 per cent are of the renal type with localization in the capillaries of the kidneys. Occasionally there are seen cases with selective localization in the pancreas, the suprarenals and rarely death may follow spontaneous splenic rupture with intraperitoneal hemorrhage.

The cerebral localization of the malarial parasites in all the fatal cases in our series suggests the possibility of a single strain of Plasmodium falciparum with an affinity for the cerebral capillaries. This is consistent with the epidemiological findings, which indicate that the disease, having been introduced by a carrier, was subsequently directly transmitted from addict to addict. In the epidemic in Egypt in 1929 and 1930 reported by Biggam[6] and by Biggam and Arafa[7] diarrhea and dysentery were prominent symptoms and apparently were caused by localization of parasites in the intestinal mucosa. Cerebral manifestations were infrequent. Diarrhea or dysentery did not occur in any of our cases.

It is apparent that a diagnosis of malaria is readily suggested and should always be considered a possibility when an obscure fever is present in a known drug addict, especially of the type using the intravenous route for injection of drugs. Such addicts usually exhibit numerous needle scars over the superficial veins in the antecubital fossae or on the arms or forearms. The possibility of malaria should also be considered in a febrile patient showing evidence of homosexuality, since drug addiction is very common in that group. The combination of fever and stupor or coma in any such person in whom drug addiction is apparent or suspected, warrants an immediate examination of the blood for malarial parasites. Stupor, coma, excitement, tremulousness, fever are symptoms which should not necessarily be considered manifestations of drug poisoning or drug withdrawal. Such interpretations were frequently given by the addicts themselves who in most instances were totally unaware of the origin of their symptoms and who in some instances considered the diagnosis of malaria either a joke or a fraud perpetrated on the addicts as a means of making them relinquish the drug habit.

Although a warning has been issued to addicts concerning the danger of malarial transmission by the common use of unsterilized syringes, new cases continue to appear but with much less frequency. During the three months period, since February 28, 1934, 5 additional estivo-autumnal cases, one of which was fatal, and 2 quartan cases were admitted to Bellevue Hospital.

The single tertian case in this series has since acquired quartan and estivo-autumnal infections, the only instance of a mixed infection in the entire group. One malarious addict, who refused to remain in the hospital for adequate treatment after his clinical condition improved, suffered a relapse and a month later applied to another hospital for treatment. Biggam[26] in 1932 reported that many addicts in Cairo, Egypt, have returned to the old habit of sniffing heroin, following the large epidemic of over 100 cases which he reported in 1929.[7] From information obtained from addicts, this return to the old habit has not occurred in New York City.

It is obviously important that carriers be effectively isolated and treated in order to prevent further direct transmission and also to prevent the slight possibility of indirect transmission of the disease to the general population by anopheles mosquitoes during warm weather. A malarial survey of all the prisons and lodging houses in various parts of the city where drug addicts congregate is indicated as a public health measure. There is also the danger of malarial carriers acting as donors in blood transfusions. Additional considerations are the possible transmission of other parasitic blood diseases such as trypanosomiasis and syphilis.

## SUMMARY

The epidemiological, clinical and pathological findings in an epidemic of fatal estivo-autumnal malaria of the cerebral type occurring in drug addicts in New York City are reported. The disease was directly transmitted from addict to addict as a result of the common practice of sharing the use of unsterilized hypodermic syringes for intravenous injections of heroin.

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[For Remainder of References see p. 146].

# COMPLICATIONS ASSOCIATED WITH THE TREATMENT OF BURNS*

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THE management of a burn case develops practical and theoretical considerations. Many ideas and outlines of the treatment of burns have been advanced but a review of the literature indicates that the greatest amount of constructive thinking and work along lines of practical therapeutics has been done in the last two decades. Many questions have been raised and it is obvious that there still remain problems for further research before we can agree on any standard form of treatment. As stated by Aldrich,[1] "Each new trend in medical progress has had some effect on the general ideas concerning burns. These new ideas were plausible at the time, but when subjected to careful analysis and experimentation, fell into disuse or were clung to out of habit or lack of a better substitute."

The responsibility entrusted to the physician treating a severe burn is not always fully realized nor appreciated by the patient or family.

Statistics[2] show that 45 per cent of the lethal burns occurring annually in the United States are in children under six years of age and in this group the outcome is often out of proportion to the severity and extent of the apparent damage done.

Since the introduction of the tannic acid treatment by Davidson[3] in 1925 a form of procedure in the care of the burn case has been set up and generally adopted. The advantages of this treatment and the results obtained by its use have been emphasized in a number of published reports.[4,5,6] Therefore the cases reported here are significant in that they indicate still a lack of knowledge as regards the proper application of this form of therapy, or at least an indifference to its proven value.

A number of patients have been admitted to the Children's Hospital with extensive burn lesions, the duration of the injury varying from several months to years. Neglect as regards the application of the proper methods of treatment, or delay in instituting such methods, has been a particularly noteworthy feature of each case history, and a factor in the success of subsequent efforts to bring about a cure. The cases reported here have been selected to represent various types of burn lesions as regards first the anatomical location, and secondly the extent and condition of the lesion at the time of admittance.

CASE 1. A white male, nine years of age, was burned over the entire left leg as the result of a gasoline fire. He was treated at home for two days and then moved to a hospital where he remained five months. The leg became infected and no improvement was noted in the local condition. Several unsuccessful attempts were made to apply skin grafts. The child's general condition became progressively worse, a severe grade of secondary anemia developed and he became markedly malnourished. He was then admitted to Children's Hospital in October, 1931.

Examination revealed an emaciated boy with the skin stretched tightly over the bones. The bony skeleton of the chest was prominent and the costal interspaces were depressed. The left lower extremity from the ankle to the hip was entirely denuded of epithelium and the granulating surface was covered with a purulent discharge.

The patient was immediately transfused with 200 c.c. of whole blood. He was again transfused four days later and pinch grafting started. The first grafts took well and the

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child's general condition improved. Three subsequent skin grafting operations were carried out at intervals of about five days. Two weeks after admittance the child developed a high fever, pain in the right costovertebral angle and pyuria. These symptoms entirely cleared up in a few days. All grafts took well and the leg was completely epithelialized and the child discharged in about two months.

Fig. 1. Case II.

Two months later the boy was readmitted with evident symptoms of ureteral calculi and this was confirmed by *x*-ray and cystoscopic examination. An operation for removal of the calculi was successful and the child made a good recovery.

About six months later there was a recurrence of symptoms and *x*-ray examination again demonstrated calculi in the right ureter. These were removed by ureterotomy and the patient made an uneventful recovery.

A plastic operation on the left knee was performed March 5, 1934 by the Keller technique to correct a contracture of the left popliteal space resulting from the burn scar. The wound became infected although the scar area previous to operation appeared clean. The graft sloughed but the area was later covered with pinch grafts and a satisfactory result obtained.

The last examination note stated that there was complete motion of the knee joint. The panniculus was good under the grafted area. There were no symptoms referable to renal or ureteral calculi.

Case II. The patient, a white male, aged four years, was admitted with a history of having been burned eight months previously when his nightgown caught fire from a burning wastebasket. He was taken to an emergency hospital where tannic acid solution was applied. The parents were told that the child could not live and he was taken home. The boy was transferred the following day to a second hospital where he remained seven days and was then moved to a third hospital where he was treated up to the time of admission to the Children's Hospital in September, 1933. According to the history, the treatment included a number of blood transfusions, saline baths, boric compresses and attempts to graft skin from the father. These attempts at grafting were unsuccessful.

The examination revealed an extremely emaciated child with an extensive infected granulating wound covering the anterior aspect of the chest, the abdomen, the thighs and the legs (Fig. 1). The posterior aspects of both thighs and legs were also denuded of skin over nearly two-thirds of their surfaces. The burned area approximated 45 per cent of the total body surface. The back was covered with large decubiti and there were deep ulcers over the trochanters of both femurs. The left hip joint was found to be dislocated.

On admittance, the child was immediately given a direct blood transfusion. Wet boric dressings were applied and on the third day following admission, skin grafting was started using full thickness pinch grafts. The chest was first covered with grafts from a small area of healthy skin along the flank. Subsequent skin grafting was done in stages—nine operations in all with grafts taken from the upper arms and from the back as the condition of the skin in this region improved. For the most part, the attempts at grafting were successful although there was a tendency for the newly epithelialized areas to break down due to diffi-

culty in controlling the infection. The child was placed on a high caloric diet, cod liver oil and malt and seven transfusions in all were given over the period of treatment.

Fig. 2. Case III.

He was discharged in April, 1934 to the convalescent hospital with all burned areas healed but with developing contractures of the right groin and right knee. His general condition had improved greatly, the weight having increased one half. A marked change in the child's disposition from a very irritable, uncooperative patient to a happy, intelligent cooperative child was a notable feature in the course of the treatment.

Note: These patients demonstrate the condition of extremis that may be present in neglected cases of severe burns. The state of malnutrition with lowered resistance to infection complicates treatment, retards healing and may result in complete failure of attempts at skin grafting. Conversely, the value of early skin grafting in extreme third degree burns employing accepted methods is demonstrated. Experience has shown that the tannic acid treatment properly carried out,[7] supportive measures and skin grafting at the earliest possible period prevent these complicated and perplexing problems that tax the ingenuity and ability of the attending surgeon. With the present established methods of treatment such cases should be a rare occurrence.

Case III. This patient, a white girl aged eight years, was severely burned when her clothing caught fire as she attempted to put wood in a furnace. She was treated in a hospital for two months and then at her home. The burned area did not heal so she was admitted to Children's Hospital in May, 1929, six months following the injury.

Fig. 3. Case III.

Examination showed a fairly well-developed female child. Over the neck and the upper chest were extensive scar contractures holding the chin fixed on the chest. Over several large areas the epithelium had sloughed leaving infected granulating surfaces (Fig. 2).

Dressings moistened with Dakin's solution were applied and six days after admittance an extensive excision of the necrotic sloughing areas was carried out over the chest, upper abdomen, neck and medial portion of the arms. Three days later, full thickness pinch grafts were applied over the upper half of the burned area. The remainder of the denuded area was grafted six days later. In five weeks, the area was entirely epithelialized and the patient

discharged. She was re-admitted for plastic operations (Fig. 3) on five subsequent occasions over a period of three years. Partial resection

Fig. 4. Case iv.

of the extensive scars and various types of grafts were employed to correct the deformities. The child has now passed the age limit and has been referred to another hospital for further treatment.

Note: This case represents a common and unfavorable type as regards treatment of burn deformities; namely, the contractures fixing the chin to the chest. It must be recognized that burns of the chest, neck and lower face almost invariably lead to extensive scarring requiring operative removal. However, strict adherence to recognized methods of treatment such as the early application of tannic acid solution, early skin grafting and the maintaining of extension of the head, influence early healing with a minimum of scarring and resulting contractures. The neglected case presents a serious and often a hopeless situation as regards a good cosmetic result. In fact, only after long and tedious

Fig. 5. Case v.

operative procedures can a satisfactory functional result be obtained.

Case iv. This patient, a white girl, six years of age, was admitted in June, 1931. Three months previous to entrance, she sustained a burn of the left arm and chest when her clothing caught fire. She was taken to a hospital and remained there several days and was then treated at home.

Physical examination revealed a large burn scar of the left chest wall adherent to the left arm from the axilla to near the elbow (Fig. 4). There was some ulceration and crusting of the scar tissue.

At operation, the adhesions of the left arm and chest were severed, allowing abduction. A broad pedicle flap with the base over the highest point of the shoulder was swung into the axilla. The scar at the elbow joint was incised and a short pedicle flap turned into the antecubital

space. The arm was held in abduction by a Thomas splint. At a second operation one week later, pinch grafts were placed over a large

Fig. 6. Case vi.

defect on the lateral chest wall and the medial surface of the arm. Epithelization progressed satisfactorily but after healing there was still scar tissue sufficient to limit motion to a considerable extent. Four months later another plastic operation on the axilla was performed, allowing full abduction. The remaining denuded areas were skin grafted later. The final result was satisfactory from a functional standpoint.

Case v. This patient, a white girl, nine years of age, admitted in April, 1931, received a hot water burn of the chest, right shoulder and right arm six years before entrance to the hospital. The treatment received at the time of accident was not recorded.

At examination, the right arm was adhered to the chest by a scar contracture forming a web 10 inches in width (Fig. 5). There was also a keloid scar extending well over the back.

At operation, it was possible to split the scar forming the web and the resulting skin flaps were sufficiently large to cover the defects and allow free motion of the arm. By means of a splint, the extremity was held in abduction while healing and when last examined three months after operation there was good movement in all directions.

Comment: These two cases illustrate the importance of position during treatment. Hospitalization would seem indicated in all cases presenting the possibility of adhesions between two adjacent burned surfaces. The difficulty in applying suitable splints and apparatus allowing satisfactory healing and avoiding or minimizing adhesions and contractures can, in most cases, only be overcome in a properly equipped hospital and under the supervision of a staff trained in the care of this type of case. A delay of six years before remedial treatment was instituted can only be classified as neglect. Nor is such delay ordinarily the fault of the attending physicians, as in most instances it is due to indifference and ignorance on the part of the parent or guardian. In fact, it is often necessary to secure a court order before such patients can be given suitable treatment.

Case vi. This child, a white female, eleven years of age, was admitted in January, 1931. The history stated that the patient was burned over the entire surfaces of both lower extremities as the result of a kerosene oil explosion. The accident occurred seven months before admission, during which time treatment was administered by a physician at the child's home.

Examination showed a markedly emaciated child. The burned area extended over both of the lower extremities and there were scar contractures at both knee joints with flexion deformities and limitation of motion.

A debridement was done with the removal of necrotic tissue to a depth of ¼ inch over the entire leg surfaces. Full extension was then possible with slight difficulty and the extremities were held in this position by splints. Skin grafting in stages was subsequently performed and satisfactory healing obtained (Fig. 6). When last seen one year later, the child walked without a limp. There was some suggestive

spasticity. Both knees could be flexed to slightly less than a right angle.

Comment: The importance of splinting or extension by means of traction during the stage of healing is demonstrated. The difficulty in combating infection is evident. The emaciation offers evidence of chronic infection and absorption of toxins from the burned areas. Early skin grafting and restraint in the proper position would have provided the best chance for early recovery and satisfactory function.

Fig. 7. Case vii.

Case vii. A white female, five years old, was admitted in May, 1930. Two months previous to admission, she was burned about the neck, chest and shoulders when her clothing ignited from a paper fire. She was treated at a hospital in another city until the time of admission.

On examination, an infected granulating area was noted extending over the right side of the neck down to the right shoulder region (Fig. 7). A marked degree of malnutrition was present.

A debridement of the infected granulation tissue was carried out and pinch grafting accomplished in stages. Several direct blood transfusions were required to improve the general condition. Complete healing was finally obtained and the child discharged home. She subsequently returned for plastic operations for the correction of contractures and removal of keloid scar. Pedicle and inlay grafts were placed and after repeated attempts over a period of three years, a satisfactory functional result was obtained.

Case viii. A colored girl, five years of age, was admitted in September, 1929. Two years before, she received an extensive burn over the arms, face and neck by flames from a stove. She was treated at home by applications of vaseline, Carron oil and picric acid over a period of three months. The healing of the burned area resulted in extensive scar contractures.

On examination, fibrous scar tissue was noted over the anterior surface of the right shoulder and on the neck (Fig. 8), extending up on the face and pulling the lower lip downward. The chin was held fixed down toward the chest (Fig. 9). There was also scarring around the right elbow.

At operation, the fibrous bands of the neck were excised and a pedicle graft was swung across the denuded area. The remaining granulating areas were covered with Thiersch grafts three weeks later. Healing progressed satisfactorily but the child's convalescence was complicated by a right lobar pneumonia and an attack of measles. Nine months later, a second plastic operation was performed to correct the remaining contractures of the neck. A satisfactory functional result was obtained.

Case IX. A white male child, nine years of age, was admitted in April, 1928. He was burned over the right leg ten months previously when his clothing ignited from a gasoline fire. There had been a discharge from the leg since the date of accident.

On examination, the right leg was found denuded of skin and covered by infected granulations from the hip to the ankle. Wet boric compresses were immediately applied and skin grafting started a few days later. The denuded surfaces were covered in stages by the pinch graft method and healing progressed satisfactorily. The child was discharged in three months with the leg healed.

Nine months later, the boy was re-admitted. The leg had remained healed for several months after discharge, when an area over the tibia ulcerated and later a small area above the knee broke down (Fig. 10). The infected granulating areas over the anterior aspect of the leg and thighs were excised, giving a clean surface which was pinch grafted one week later. The grafts took well and the patient left the hospital in one month with epithelization complete.

Fig. 8. Case VIII.

Fig. 9. Case VIII.

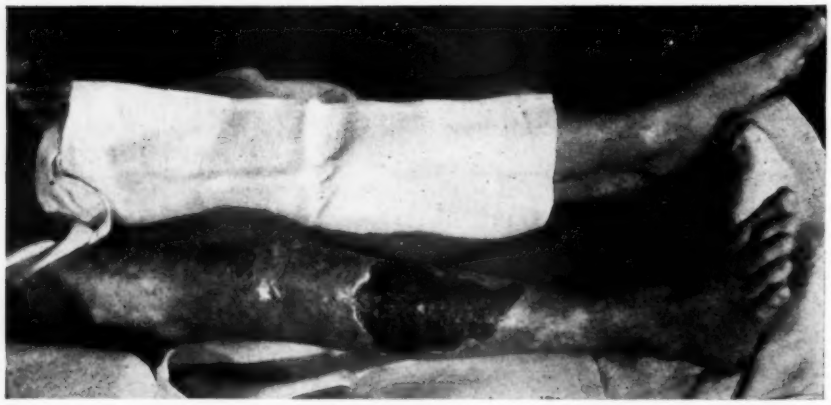

Fig. 10. Case IX.

Case X. A white, female child, three years of age, received burns about the neck and body four months before admittance when her clothing caught fire while she was playing with matches. She was treated in another hospital for ten weeks and then admitted to Children's Hospital in January, 1930 for a plastic operation on the neck.

On examination, there were many healed scars extending over the anterior portion of the neck and the right side of the chest and back.

There were also smaller scars on the abdomen, right leg and right arm. There was some limitation of motion of the right arm and the chin

Fig. 11. Case x.

was held fixed on the chest (Fig. 11).

A plastic operation was done on the neck, excising the scar and swinging two pedicle flaps over the denuded area, one from each side. Two weeks later the remaining denuded areas were covered with pinch grafts. The areas healed fairly well and the child was discharged three weeks later.

Three months later, the patient was readmitted and three subsequent operative procedures were carried out to correct the contractures of the neck. Healing was complete in two months and the child was discharged.

She returned to the hospital three and one-half years later with a partial recurrence of the scar contracture of the neck. The deformity was corrected by means of a z-type plastic operation. Healing was prompt and the immediate functional result satisfactory.

Case xi. This patient, a white female child, eleven years of age, was admitted in March, 1934. The history stated that she sustained an extensive burn of the back nine months previously when her dress ignited from a bonfire. She was treated with tannic acid spray with at least one daily application for a period of four months. No history of any other type of treatment was obtained.

Fig. 12. Case xi.

Examination revealed a moderately emaciated child. There was an infected granulating wound over the upper half of the back and left shoulder. The right upper arm was adherent to the chest wall for one-half the distance to the elbow (Fig. 12). The exuberant granulation tissue had a noticeably unhealthy appearance and was covered with a profuse purulent exudate.

Dressings wet with Dakin's solution were applied alternating with exposure to lights. Pinch grafting was begun four days after admission. The results were rather poor with a small percentage of takes probably due to the extensive infection. Subsequent pinch grafting has been partially successful and the wound is slowly healing.

Comment: The foregoing case record demonstrates that at least a part of the medical profession lacks the knowledge sufficient to successfully treat the severe

burn case. A child with a serious burn is allowed to remain in a state of chronic invalidism and develop a dangerous infection as the result of inefficient treatment extending over a period of nine months. The resulting deformity developing from adhesions and contractures provides a difficult and perhaps insurmountable problem for the surgeon. The unnecessary physical and mental trauma that the child has been forced to endure provides the greatest argument for the adoption of proper treatment in all such cases. We believe that this treatment is best provided by the trained surgical staff and properly equipped hospital.

## SUMMARY

A group of cases is presented illustrating the result of what may be considered maltreatment of burn lesions. Each case has been selected to emphasize this factor as evidenced by the application of improper or incomplete methods of treatment. Attention is called to the long periods of morbidity, the local and general conditions as presented at the time of admission and the difficulty in obtaining a satisfactory end-result by subsequent treatment.

The fact that legal procedures have not been instituted is perhaps a matter of good fortune. Strict adherence to the accepted principles of treatment as proposed by Davidson and others will, we believe, in the majority of cases prevent the unfortunate occurrence of these all too common complications. Prompt application of local treatment, proper supportive measures, follow-up care and attention, together with the correction of deformities as indicated offer a reasonable hope for success. These unfortunate victims are deserving of the best care the attending surgeon can provide. The satisfaction derived from such work well done and carried to a successful termination is a worth while reward.

## CONCLUSIONS

1. Numerous ideas and outlines of the treatment of burns have been advanced and many questions have been raised indicating the need for further study.
2. A number of maltreated burn cases have been observed at the Children's Hospital during the past few years.
3. The treatment of such cases presents complications and the results are often unsatisfactory.
4. Certain cases demonstrate a lack of knowledge of accepted methods of treatment.
5. In certain instances, indifference on the part of parent or guardian is apparent.
6. The treatment of extensive burns is best carried out in the well-equipped hospital under the supervision of a staff trained in the care of these cases.

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# TRAUMATIC INTRACRANIAL HEMORRHAGES*

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## I. CONTUSION AND LACERATION OF BRAIN

UNDER modern conditions injuries to the head have become so common and the fatalities following these injuries so frequent that I have commented facetiously that the average American was in greater need of a head protector today than was the average member of the American Expeditionary Forces in France in need of a trench helmet. In my series of automobile deaths alone injuries to the head were responsible for 45 per cent of the deaths.

While it is true that a high percentage of these deaths was inevitable it is also true that in a considerable percentage intelligent operative intervention offered at least a certain amount of hope. In brain tumors operative intervention is by election, with opportunity for the leisurely carrying out of every step in the surgical toilet of the brain. Intervention in relation to head injuries often requires a more rapid emergency technique. In general the modern laissez-faire attitude toward skull fractures together with lumbar drainage has in large part justified itself. The superimposing of operative shock upon traumatic shock was responsible for an inexcusable mortality a generation ago when every case of head injury tended to come to immediate operation. The picture of lowered mortality in reported series of skull fractures under modern conditions has been due in part to the inclusion of minor *x*-ray cracks of the convexity apparently with little or no damage to the brain. This group of minor cracks of the convexity practically never gives rise to symptoms which would suggest surgical intervention. Among the greater group with more extensive damage to the brain occur cases in which the patients survive the traumatic shock but do not come back to normal, symptoms persisting which suggest the presence within the skull of hemorrhages which are compressing the brain. In this group operative intervention after the traumatic shock has abated is often of great value.

This series of type cases is not offered in criticism of the failure to diagnosticate the conditions. Our knowledge in this field is limited, the diagnostic signs and symptoms are not always clear, the emergency conditions under which these cases are often seen are confusing and not conducive to quick judgment which the situation requires. The problem in many cases is put directly on the doorstep of the general surgeon, who is on the spot. Delay for consultation with neurosurgeons from a distance may be fatal. The development of an exploratory approach has tended to clear the atmosphere in cases where there is clinical evidence of intracranial hemorrhage but no focal signs indicating on which side pressure was being brought to bear.[1] Fortunately in the extracerebral hemorrhages whether epidural or subdural the blood or clot tends to accumulate over the cerebral convexity on one side and to be located where a subtemporal burr hole will usually disclose its presence. A perfected operative technique which will lead to quick "get in and get out" procedure, with due

[1] Munro, D. The diagnosis and treatment of subdural hematomata. *New England J. M.*, 210: 1145, 1934.

* From the Medical Examiner Service, Suffolk County, Southern District, Boston.

consideration of asepsis, promises best results.

A case illustrating the successful outcome of late operative interference in a fractured skull is the following:

Case 1. No. 19145. Male, thirty-four years of age. Fell down stairs in a subway station March 23, 1917. He was admitted to the surgical service of the Boston City Hospital semiconscious, with an odor of alcohol on his breath. Pupils were equal and reacted to light. He was bleeding from the left ear. Reflexes were abolished. Some hours after entrance he became restless and required restraint. The following day, March 24, alcoholic symptoms disappeared; his mental condition improved somewhat; he answered simple questions rationally. He moved his right arm and leg more than the left. Neurological examination March 25 disclosed paresis of the left face, arm and leg; the right knee jerk less than the left; Babinski and Oppenheim reactions on left. Eye examination disclosed normal fundi. The pupils were equal and reacted. He was dull, irritable and confused. March 26 he could move the left arm and left leg well. Right knee jerks were greater than left; Babinski reaction still present on left. He preferred to rest on his left side. March 31 he had had periods of relief, mental clearness being followed by dullness and confusion. The left arm was partially paralyzed, otherwise the condition was unchanged. On the evening of March 31 he became comatose. The left arm and leg became totally paralyzed. Dr. Arthur R. Kimptom performed a right subtemporal decompression which disclosed no epidural hemorrhage. On opening the dura there was an escape of considerable old blood clot followed by fresh bleeding controlled by warm salt solution. The brain returned to its normal contour. The skin was closed without drainage. He was discharged April 27, 1917, mentally clear and oriented with no abnormal neurological signs.

He continued to drink for a time but following what appeared to be an epileptic seizure December 15, 1918, he reformed.

His history from this point is that of a useful citizen. He ultimately became receiving clerk for an important variety store occupying a whole business block. This work required mental acumen and alertness. He was very efficient, but had an occasional epileptic seizure. On September 25, 1929 he had such a seizure and fell striking his head against a projecting corner of a desk base. He was unconscious for a few minutes. When consciousness was restored he became clear mentally but complained of dizziness. He was taken home in a taxi and lay down on a bed. After a short time he went to sleep. His sleep became progressively deeper and after about two hours he could not be roused. His breathing became progressively more noisy. A local doctor was called who ordered him into a hospital. On admission he was unconscious with fixed pupils of normal size; respiration was stertorous. There was froth in the mouth. Limbs were rigid. Lumbar puncture produced a blood-tinged fluid. The heart became irregular. Intravenous luminal led to relaxation of the rigidity but the heart became rapid and he died twelve hours after the accident and six hours after admission to the hospital.

Postmortem examination disclosed a recent fracture arising 2 cm. in front of the left parietal eminence and proceeding forward and downward, crossing the squamous bone and then across the base in the left middle fossa to terminate in the foramen rotundum. In its course the fracture followed the groove for the posterior branch of the middle meningeal artery for 2.5 cm. Separating the dura from the skull in the left temporo-parietal region was a massive epuidural clot, 11 cm. from before backward, 10.5 cm. from above down and 4.5 cm. in thickness in its mid region (Fig. 1). The brain was deeply compressed by the clot on the left and flattened against the skull on the right.

Marking the end results of the 1917 injury, on the right side a window of fibrous tissue and dura occupied the site of the operative opening of the skull in 1917. Adhesions between dura and brain were present over the basilar surfaces of the right frontal and temporal lobes back to the mid portion of the temporal lobe. Over the convexity adhesions were present over the inferior half of the right frontal lobe, over the anterior portion of the right temporal lobe and the adjoining parietal lobe. The anterior portion of the right temporal lobe was occupied by a series of cysts save for a region 1 cm. in thickness along the basilar junction (Fig. 2). Regions of loss of cortex and shallow cyst formation were present along the basilar surfaces of both frontal and temporal lobes with similar patches over the lower convexity of right frontal lobe.

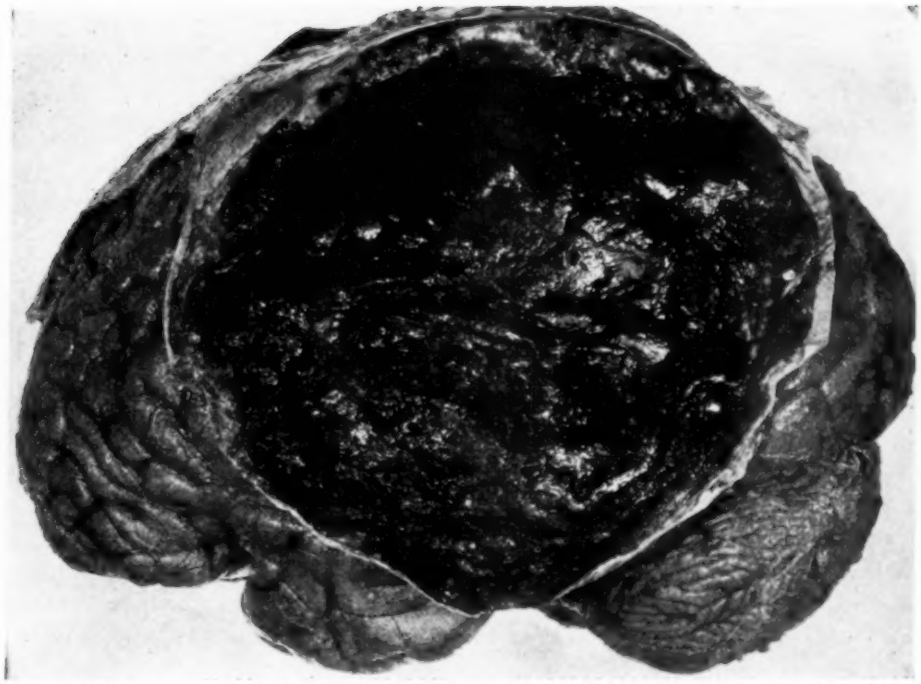

FIG. 1. Massive extradural hemorrhage. Narrow layer of dura is seen about edge of clot.

FIG. 2. Traumatic cyst of brain and adhesions of dura. Dural window through which decompression was performed is indicated by pins thrust through it.

*Comment.* This case illustrates: (1) The common occurrence of epilepsy following adhesions of brain and dura due to the repair process. The complete mental recovery save for epileptic seizures at long intervals and the ability to carry out relatively complex work are notable. (2) The proneness to second head injuries as the result of falls. (3) The failure to diagnosticate an extradural hemorrhage with a classical symptom complex. This was overlooked in a general hospital with a very efficient staff, perhaps because of the emergency conditions. Knowledge that the patient was an epileptic may have led to confusion since his condition may have been looked upon as due to a post-epileptic state. The subdural hemorrhage was the largest and thickest the writer has ever seen. Perhaps compression of the cystic portions of the right lobe allowed for the accumulation of an unusual amount of blood before the intracranial pressure reached the fatal degree.

### II. EPIDURAL HEMORRHAGES

It would seem to be unnecessary at this time to call attention to the mechanism producing extradural hemorrhage and its characteristic symptom complex. However it is not uncommon in our medical examiner service in Boston to find at postmortem examination epidural hemorrhages which have been overlooked clinically, as in Case 1. Dr. Milton Helpern, assistant to Dr. Charles Norris, chief medical examiner in New York City, informs me that their experience corresponds to mine. The dura is a simple fibrous membrane which is readily peeled from the skull over the lateral convexity, except in the very young and the old. As it is peeled from its attachment there come into view the branches of the meningeal arteries, which stand out as if embossed on the outer surface of the membrane, and lie in shallow grooves on the inner surface of the skull. When one remembers that the squamous portion of the temporal bone is the thinnest and most brittle portion of the convexity of the skull one can understand how relatively slight blows in this region can produce fractures which tend to cross or follow the grooves for the meningeal arteries. Dislocation and rupture of the artery tends to follow. The blood has to strip the dura from its attachment as it collects and overcome intracranial pressure. Moreover the roughened surface thus exposed favors rapid clotting, so that the accumulation of blood is slow. There is usually no associated damage to the brain and its covering arachnoid. While the accumulation of blood is slow it is usually continuous since the bleeding is from one of the larger branches of the middle meningeal artery which cross the squamous bone.

There is usually temporary unconsciousness, due to the fall. This is recovered from and the individual appears to be none the worse for the injury, save perhaps for slight headache or dizziness. Then after a period, often of hours, he becomes sleepy. As the blood accumulates and the intracranial pressure increases the sleep deepens into semi-coma and coma is followed by death, unless surgical aid is obtained. One of the nearest approaches to a surgical miracle results from successful surgical intervention in this type of hemorrhage.

The following case illustrates a failure to recognize a somewhat atypical epidural hemorrhage, in which there is a suggestion that the hemorrhage ceased for a time to be later reestablished.

Case II. No. 24458. A boy, five years of age, was coasting in a play cart when the cart overturned, throwing him to the ground. He was temporarily unconscious. He was seen by a local doctor some hours later. There was a hematoma of the left temporal region. The child was somnolent. He vomited once during the night. He appeared to be better the next morning but was sleepy in the afternoon. On the next morning he developed projectile vomiting and became unconscious. A second doctor was called in. *X*-ray disclosed a fracture of the left convexity of the skull from the parietal

eminence forward and downward crossing the squamous bone. With another physician a lumbar puncture was performed. A clear normal fluid was obtained. The child regained consciousness shortly after the lumbar drainage. On the next morning he was conscious and rational. Later in the morning he became cyanotic and lapsed into unconsciousness. Lumbar puncture again obtained a clear fluid but without relief of symptoms and the child died fifteen minutes after the puncture.

The postmortem examination disclosed a hematoma of the scalp in the left temporo-parietal region 4 cm. in greatest diameter. The dura was separated from the skull in the temporo-parietal region by a mass of clot with some fluid blood 7 cm. in diameter and 1.5 cm. thick in its mid region. The brain was flattened by the epidural clot, but was otherwise normal. A fracture arising below and behind the left parietal eminence proceeded forward and downward into the base of the left middle fossa terminating in its mid region. In its course it crossed grooves for two of the posterior branches of the middle meningeal artery and near the junctions of convexity and base crossed the groove for the main vessel.

*Comment.* There are several lessons to be learned from this case. (1) The hematoma was located in a region likely to be associated with a fracture crossing the grooves for the branches of the middle meningeal artery. (2) The finding of a clear lumbar puncture fluid in a case with a history of a fall followed by temporary unconsciousness—to be in turn followed by somnolence, vomiting and unconsciousness, should have suggested that there was no damage to the brain or arachnoid, and that the pressure symptoms were probably due to a hemorrhage outside of these structures, i.e. either subdural or epidural. (3) The temporary relief of symptoms following the first lumbar puncture was confusing to the doctors in the case because they reasoned that it had accomplished good and could be repeated if necessary, probably with repeated relief. As a matter of fact it did let up on the intracranial pressure temporarily, but probably furnished a basis for a reactivation of the epidural bleeding. (4) Much was made by the doctors of the fact that there were no paralyses. It is unusual for an epidural hemorrhage to produce any paralysis, except perhaps *rarely* a temporary hemiplegia. The pressure of the blood tends to be so diffused as it accumulates that focal pressure over the motor area adequate to produce paralyses does not arise.

### III. SUBDURAL HEMORRHAGE

This supposedly rare condition is relatively common in my experience.[2] Because of the peculiar conditions which exist in the subdural space the burden of organizing a subdural clot is thrown on the relatively avascular dura. The poor circulation, notably the lack of adequate provision for venous drainage of the granulation tissue, and the necessity of organizing the clot from only one surface, the dural, are responsible for the poorest effort at repair found in the human body. Months and sometimes years elapse before the process is healed, if ever. Intervening inflammatory processes add complexity to the microscopical picture, and are responsible for the name pachymeningitis hemorrhagica interna chronica which Virchow applied to late stages in the picture. The chronic passive hyperemia which prevails in the repairing process and the poor supporting tissues which are produced lead to wide dilatation of the capillaries of the granulating layer. These "giant capillaries" tend to rupture and produce secondary hemorrhages which renew at intervals the burden thrown upon the dura in its efforts at repair. They may lead to such increases in intracranial pressure as to cause dramatic clinical manifestations in the course of the disease, or may cause death if the increase in intracranial pressure is rapid and high enough.

Subdural hemorrhages in the infant are usually due to birth injuries frequently to the tentorium. In the adult such hemor-

[2] Leary, T. Subdural hemorrhage (reporting 50 cases). *J.A.M.A.*, in press.

rhages may be due to contusion and laceration of the brain, incident to or independent of fracture of the skull. Most of the hemorrhages which occur independently of fracture in the adult are of venous origin, arising from rupture of bridging veins which cross the subdural space, or of veins on the surface of the arachnoid. This type usually follows mild traumatism, often in the form of minor falls, sometimes falls in which there is no direct impact to the head. Alcohol is an etiological factor in 40 to 60 per cent of the cases.

A study of the subdural space has demonstrated that it is not a serous space comparable to the pericardium, pleura and peritoneum.[3] The most reasonable concept is that the skull, like other bones, enters into articulation. In this case the articulation is with the brain and its highly impermeable covering, the arachnoid, which has no capillary bed. The dura from this standpoint is the lining of an articulation, and as such is made up of simple fibroblastic tissue. When a hemorrhage occurs into the subdural space the impermeable arachnoid plays no part in the removal of the blood. As explained above the relatively avascular dura is not competent to efficiently organize and remove the blood. A layer of poor granulation tissue invades the clot from the dural surface and a thin enveloping layer of fibrous tissue is slowly formed which separates the clot from the arachnoid. The blood undergoes slow hemolysis and liquefaction within the neomembrane. Liquefaction is associated with the breaking down of protein molecules, leading to increased osmotic pressure and absorption of fluid by dialysis, with resulting increase in bulk within the neomembrane and increasing intracranial pressure.

Histiocytes carry blood pigment from the broken-down corpuscles into the arachnoid where they give it up to the cerebrospinal fluid. As a result the fluid is stained yellow, i.e. xanthochromia is produced, which is an important sign, if present. It is needless to say that the subdural space is a closed space. Lumbar puncture cannot drain this space. The only evidence presented by the cerebrospinal fluid is a possible xanthochromia and perhaps lymphocytosis.

Unfortunately the symptom picture is often not very clear. A history of injury followed by headache, mental changes, often of minor character but sufficiently definite to be recognized by the family, and frequently associated paralysis of one facial nerve should suggest a subdural hemorrhage. Dilatation of one pupil is a not rare finding. Persistence of these symptoms should indicate the desirability, and increase of symptoms the necessity, for exploratory intervention. The process is unilateral in about 80 per cent of the cases unassociated with fracture. In my series there was clinical failure to recognize or even suspect subdural hemorrhages in all of the cases arising outside of hospitals, and frequent failure within these institutions until a recent period.

The following cases illustrate clinical and pathological findings in well-studied type cases in which the diagnosis of subdural hemorrhage was not made.

Case III. No. 22708. A man, aged forty-seven years, was working on a platform about 3 feet above the level of the floor December 14, 1932, when there was a flash as electric power was turned on at 11 A.M. He either jumped or fell to the concrete floor which was oily and slippery. He picked himself up and walked to the plant hospital. He was not unconscious and showed no oil on his hair, having apparently fallen without striking his head, but with an abrasion of the right elbow and a contusion of the left knee. He was said to have been drowsy and to have gone to sleep in the shop when not working during the week preceding the accident. He had been a heavy drinker at times. He remained in the plant hospital until 4 P.M. on the day of the accident. He was then taken home. The next morning he was very nervous, showed tremor of the hands, was

[3] Leary, T., and Edwards, E. A. The subdural space and its linings. *Arch. Neurol. & Psychiat.*, 29: 691 (April) 1933.

mentally clear and speech was clear. December 16 the left pupil was larger than the right. He walked to an ambulance, favoring his right leg, and had a slight limp. His sister reported that he had not slept on the nights of December 14 and 15. He was very restless in the hospital and at 1 A.M. became disoriented. Disorientation was evident from this time, particularly at night. He became incontinent. On December 17 there was paralysis of the right arm and leg. On two occasions later the urine was of high specific gravity and contained 0.2 per cent sugar. Other tests of urine and blood were negative, including a Wassermann. At the hospital *x*-ray examination of the skull was negative. Lumbar puncture disclosed a clear fluid with one cell and the fluid was under normal pressure. A surgical consultant concluded that there was nothing surgical in the picture. A medical consultant was called whose physical examination was negative, save for partial paralysis of the right arm with a weakened hand grip. The right shoulder was tender to the touch. The medical consultant concluded: "It is my opinion that from the paralysis, the presence of sugar in the urine, with the blood sugar normal, and the lack of confirmatory evidence by *x*-ray of trauma to the skull, combined with a non-bloody spinal fluid, that this man suffered from a hemorrhage in the hind part of his brain, independent of any injury, and it was this hemorrhage which caused his dizziness and subsequent fall from the platform."

An eminent neurologist examined the patient, whom he found restless and disoriented. The left pupil measured 0.4 cm., the right 0.2 cm. Pupils were regular and reacted to light. There was slight internal strabismus with no evidence of paralysis of the eye muscles, the eyes moving freely in all directions. The tongue did not deviate. There was weakness of the muscles of the lower face on the right side. The eyelids were closed firmly. The right arm was moved little voluntarily; was moved when he was urged, but not above the level of the mouth The right leg was less strong than the left There was moderate spasticity of the right arm and leg. The right triceps and biceps reflexes were greater than those on the left. Knee and ankle jerks were unsatisfactory because the leg muscles were not relaxed There was a distinct extensor response to the plantar reflex on the right but no Oppenheim or other abnormal reflex. No differences in sensory reactions could be obtained between the two sides of the face and body.

The examiner concluded: "This man has evidently had a thrombosis of the left middle cerebral artery, probably coming soon after the fall. This is shown by the paralysis affecting the face, arm and leg, all in nearly the same degree though the arm is most affected, as it generally is when the motor fibres are affected at or near the internal capsule. A hemorrhage from this artery would cause the same distribution of the paralysis, but is accompanied by more severe symptoms at the onset, and almost invariably by unconsciousness. Hemorrhages from a cerebral injury are not apt to be so deep in the cerebral substance, and so cause paralysis that is not so widespread, and this again is almost invariably accompanied by unconsciousness, and often by convulsions at the onset, and the paralysis appears at once and not after an interval. Rarely a cerebral hemorrhage may appear late after a cerebral concussion, but then the interval is longer, a week to three or four weeks, and there is almost invariably unconsciousness at its onset, and generally also at the time of the injury, or at least well-marked signs of cerebral concussion at the time of the injury which were absent in this case. I can come to no other conclusion than that the thrombosis in this case was merely coincident with this accident, and that there was no cerebral injury at that time, nor anything more than a mental shock with perhaps some feelings of weakness and shakiness afterwards, but nothing which could by any possibility cause a thrombosis in a cerebral vessel. This is undoubtedly due to the usual causes at the age of this man, namely a roughening of the inner coats of the vessel wall, and a clotting of the slowed blood stream, when the person is at rest as during sleep."

The patient was transferred to the medical service of a general hospital December 27. He was stuporous. The right pupil was small but reactive to light The left pupil was moderately dilated and fixed to light. Fundi were negative except for very small constricted arteries. The neck was stiff, lungs emphysematous, heart regular in rate and rhythm. Peripheral vessels were moderately thickened. B.P. 190/120. There was slight right facial weakness. Spastic musculature on left. Right biceps and triceps jerks less than left. K.J. and

A.J. active and equal. A white count of 23,400 at time of admission fell to 15,000 and to 11,000 just prior to death, with pre-

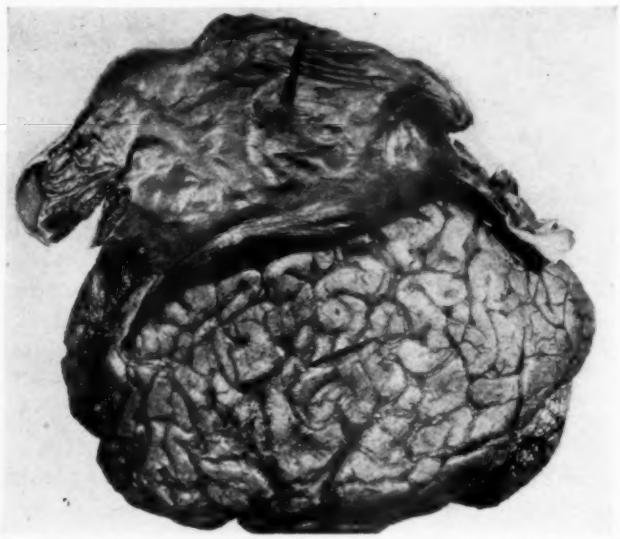

Fig. 3. Subdural hemorrhage with formation of double neomembrane. Internal thin layer of membrane is seen, with fragments of clot within membrane visible at top and at left edge. Wrinkling of inner layer is due to loss of fluid. Molding of brain by pressure is evident.

dominating polynuclears. Urine essentially negative. N.P.N. 43 and 44 in two tests. Blood Kahn reaction negative. Lumbar puncture revealed a clear fluid with pressure of 200 mm., normal dynamics, no cells, a total protein of 21, negative colloidal gold reaction, and a negative Wassermann. The patient was stuporous and then restless requiring restraint. The evidence of hemiplegia failed to clear during his stay in the hospital. There were definite signs of a terminal bronchopneumonia. Death occurred January 4, 1933. The discharge diagnosis was "Hypertension. Cerebral hemorrhage or thrombosis. Bronchopneumonia."

Postmortem examination of the head disclosed no evidence of injury to the scalp and skull. The dura over the left convexity was discolored greenish brown. On opening the dura the scissors entered on the left side a cavity from which there was an escape of a large amount of brownish bloody fluid (Fig. 3). The dura then collapsed over a markedly flattened left cerebrum. The dura was lined on this side by a thick layer of chocolate clot partly organized. Internal to this was the cavity from which the fluid escaped. This cavity was walled off from the surface of the arachnoid by a paper-thin internal membrane to which some chocolate clot adhered. The neomembrane enclosing the clot and fluid extended from the anterior frontal region backward to the cuneus and from the mesial junction of convexity to a region 1 to 2.5 cm. from the junction of convexity and base. The brain was compressed in the mid region to a depth of 2 cm. Surrounding the mass of clot and fluid the neomembrane was fused and less than 0.1 cm. in thickness. Adherent to the dura on the right side were small patches of pigmented neomembrane less than .05 cm. in thickness.

*Comment.* This patient was seen by a general practitioner, an internist, a surgeon and a neurologist; was treated for nine days in a medical service of a general hospital. At no time was subdural hemorrhage even considered in the diagnosis.

Case IV. No. 21747. Male, fifty-one years old, salesman. He formerly drank but had not had any alcohol for months. On the morning of February 10, 1932, while on his way to the garage in the rear of his house he fell heavily on the ice, striking on his left elbow and thigh. He did not strike his head and was not unconscious. He felt somewhat shaken, but drove his car several miles into Boston and went about his business. Three hours after his fall while showing his material in a store he became dizzy

and nauseated; he couldn't see well and "felt tough." He went across the street to a drug store, thinking that a bromoseltzer would help him, and collapsed in the store. He was admitted to a hospital unconscious. His unconsciousness lasted about thirty minutes. After recovering consciousness he was lethargic and confused, but shortly became oriented. The right pupil was slightly larger than the left, but both reacted. Extra-ocular movements were normal. There was relative hyperactivity of the deep reflexes of the left arm and leg. Temperature was 97°F. He developed a slight generalized tremor of the body, especially after being exposed to the air for a few moments. This subsided in a few hours. He complained of left-sided headache. Lumbar puncture showed an initial pressure of 170, dropping to 95 after removal of 7 c.c. The fluid was slightly cloudy and pink with 11 lymphocytes, 2250 red cells, and 68 mg. protein per 100 c.c. A provisional diagnosis of fractured skull or edema of the brain was made. *X*-ray disclosed no evidence of fracture.

Nothing remarkable marked the record, save for slight headache after reading, until February 16 when he had a severe headache. February 17 lumbar puncture showed an initial pressure of 225; 9 c.c. were removed with a final pressure of 100. The fluid was slightly xanthochromic with 45 red blood cells and 18 lymphocytes. Lumbar puncture on the 19th showed an initial pressure of 215. The removal of 8 c.c. dropped the pressure to 95. Fifteen lymphocytes were found. The headache improved after the punctures. There were slight hyperactivity of the reflexes on the left and an equivocal plantar response. Complaint of headache continued. Lumbar puncture on the 20th showed an initial pressure of 170; 4 c.c. were removed with a final pressure of 85. The fluid was slightly xanthochromic with 24 lymphocytes. February 22 the patient became somewhat stuporous and hard to arouse. Lumbar puncture revealed a pressure of 75. No fluid was removed. There was some difficulty in swallowing and speech was moderately thick. He became comatose on February 24, was fed nasally and by clysis. The temperature which had been normal became 100°F. and he became incontinent. The lungs filled up and he died with respiratory failure February 25. The final clinical diagnosis was "Edema, congestion and contusion of brain.(?) Subdural hematoma. Cerebral hemorrhage with softening. Mild upper respiratory infection."

Fig. 4. Subdural hemorrhage with formation of outer layer of neomembrane only. Naked rough clot is seen attached to inner surface of dura.

Postmortem examination of the head revealed (Fig. 4) a thick layer of chocolate brown

clot adherent to the dura on the left side, with an abundant brownish fluid lying internally in contact with the pia-arachnoid. The clot was not confined on its internal aspect by a limiting membrane save for small regions near the edges. The clot covered the whole of the convexity save for the anterior frontal convolutions and a part of the cuneus. It extended as a thinner layer onto the base over the middle fossa and the upper surface of the tentorium. There was a thin smear of slightly brown blood, not adherent to the dura, over the right convexity in a zone 2 to 3 cm. wide near mesial junction. There was no evidence of traumatism to scalp, skull or brain. The vessels at the base of the brain were very mildly arteriosclerotic.

*Comment.* This case had the advantage of careful study in a very efficient neurological service. Subdural hemorrhage was thought of casually in the final diagnosis, but was not given serious consideration in spite of the xanthochromia.

## SUMMARY

Type cases are here presented of extracerebral intracranial hemorrhages which escaped diagnosis by men who should have been more expert than the general practitioner of medicine. In 3 of the 4 cases diagnosis was faulty at the hands of men and services supposed to be highly expert in neurological diagnosis. As indicated above the cases are published in an effort to be helpful and with full recognition of the difficulties in diagnosis, particularly in subdural hemorrhage. The opportunity for obtaining exact information by a relatively minor operation, the exploratory burr hole procedure, which Munro and his associates have developed at the Boston City Hospital, should serve to clear up some of the mysteries in a neurological field in which exact information has been lacking.

When it is remembered that the only treatment of extracerebral intracranial hemorrhages is operative, and that the results of successful operation are brilliant, the necessity of using every means to perfect the diagnosis in postraumatic cerebral disturbances is obvious.

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* Continued from p. 123.

# SO-CALLED SPONTANEOUS SUBARACHNOID HEMORRHAGE

## A RESUME WITH ITS MEDICOLEGAL CONSIDERATION

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THE subject Spontaneous Subarachnoid Hemorrhage might seem out of place in a surgical journal. Certainly it has little interest from a standpoint of active surgical procedure, but it should be of general interest as it must be differentiated from traumatic subarachnoid or subdural hemorrhage. It may be a cause of sudden death and it does have a medicolegal importance. Clinically, it is a syndrome characterized by precipitous, sudden onset, with extreme violent headache, dizziness, nausea and collapse. There is marked rigidity of the neck and a bloody spinal fluid. There may be partial or total loss of consciousness but there is no gross cerebral localization. It constitutes, according to Martland, about 2 per cent of sudden deaths and, although occasionally a death may occur within an hour, it is never instantaneous. Probably 50 per cent of patients recover. This condition is much more common than is generally realized and its diagnosis is often missed, chiefly because it has not been thought of. It is comparatively a very recent addition to our list of diagnoses and, although many cases are now being reported, it is still not generally well understood.

### ETIOLOGY

It is the belief of the writer that spontaneous subarachnoid hemorrhage is almost always the result of bleeding into the subarachnoid space from a rupture of a preexisting aneurysm in or contiguous to the circle of Willis. Hence in his mind, its terminology is rather an unsatisfactory one, as the qualification "spontaneous" suggests a lack of knowledge of its exact nature and connotes a most unusual meningeal pathology, such as diapedesis, oozing from cerebral veins or some peculiar vasomotor states. Cases have also been considered as hemorrhagic encephalitis or other unusual neuropathy. These, the writer believes, are erroneous concepts, as such presumptive pathology could hardly produce the characteristic violent, sudden onset which so logically indicates the mechanical spurting extravasation of a sharp hemorrhage. At autopsy these aneurysms may be often difficult to find as they may be well embedded in a surrounding blood clot. Sometimes they are "blown out" or disintegrated, (Fig. 1) so if they are not carefully looked for they may be easily missed; undoubtedly this has occurred.

In a series of 64 cases to be reported by the writer elsewhere in each of 14 cases diagnosed clinically and coming to autopsy an aneurysm was found. It is suggested that a better and more definite nomenclature would be "subarachnoid hemorrhage from probable rupture of an aneurysm of the circle of Willis." However, it would seem that the present term is here to stay, but the confusing qualification "spontaneous" must bring to mind the fantastic theories of spontaneous generation applied to fermentation, putrefaction and fevers before the days of Pasteur and Koch. It must be understood that subarachnoid hemorrhage from trauma and from extension of an intracerebral hemorrhage is excluded under the classification of "spontaneous."

It is true that spontaneous subarachnoid hemorrhage may occur with blood dyscrasias, as in purpura, hemophilia or leucemia, and one case has been described

by the writer as a probable result of human serum reaction. It may also occur with cerebral neoplasms, especially the angiomata. It has been described as a result of sunstroke, but this, too, is considered erroneous. We then repeat our conception that spontaneous subarachnoid hemorrhage is the result of a rupture of the circle aneurysm and any presentation of this subject must include, as an important part, the careful consideration of the cerebral aneurysm.

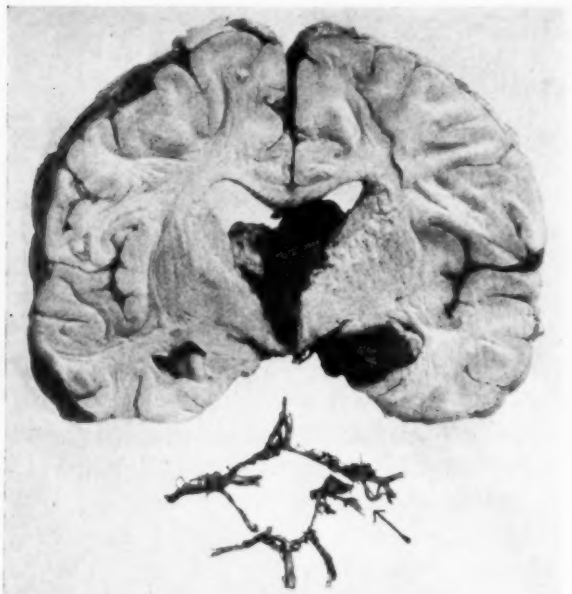

Fig. 1. Fragmented "blown-out" aneurysm of left internal carotid in a forty-five year old housewife. Two episodes of leaking thirty and ten days before fatal rupture. Such an aneurysm might readily be missed.

### THE CEREBRAL ANEURYSM

Cerebral aneurysms have been described for a great many years. Gull[1] in 1859, Charcot[2] in the 60's, Bramwell[3] in the 80's, and Fearnsides[4] in 1916, have written extensively upon them. Bramwell and Gull both remarked their occurrence in vessels otherwise free from disease. They frequently occur in young individuals. These aneurysms are usually small, so-called "berry" aneurysms averaging less than 1 cm. in diameter and occurring at junctional points in the circle or polygon of Willis. They seldom attain to a larger size without rupture; they may do so and may then present with manifestations of tumor. In the author's entire series of 20 autopsy cases only 3 were of large size, averaging 4.5 cm. in diameter.

In a discussion of the probable causes of this aneurysmal formation, the ordinary arteriosclerotic or atheromatous degeneration is, of course, a common and evident cause in older persons, but it is surprising that there is rarely a direct tear or rent in such sclerotic vessels. However, there is well over 50 per cent of cases in which there are no such demonstrable accompanying vascular changes and such aneurysms, especially in younger people, have been ascribed by exclusion to an hypothetical congenital defect. This, in the mind of the writer, is not entirely a satisfactory explanation. It is a well-known fact to every pathologist that the circle of Willis is especially prone to anomalies. Its vessels vary in size and arrangement and in such an anomalous circle there might well be false budding, forming a true congenital aneurysm, that is, an aneurysm present at birth. Cases occurring in members of the same family have been described and the writer reports two occurring in maternal cousins of six and ten years respectively. These most probably are congenital. On the other hand, it is believed that many aneurysms occurring on apparently healthy basal vessels may be simply mechanical developments due to the special stress and strain to which this remarkable distributing apparatus is subjected.

### CIRCLE OF WILLIS

Here it might be well to review the arrangement and structure of the circle of Willis. It is a most unique anastomotic circuit with marvelous potentialities for collateral supply. It is really suspended in the large fluid space at the base of the brain; it is unaccompanied by veins and lacks the surrounding structural tissue support characteristic of vessels elsewhere.

There is undoubtedly some hydraulic protection by the surrounding mobile spinal fluid, but this cannot be comparable to that of the fixed tissues. The vessels are especially thin-walled, as are the intracerebral vessels; their tenuity is due to a lack of media and elastica. At autopsy these arteries are collapsed and do not hold their contour as do peripheral vessels of similar calibre. They appear especially thin and transparent, almost like cellophane. Another point of considerable importance is the angulation of flow, as these vessels, at junctional points, produce almost right-angled channels that must bear the brunt, especially at the emergence of the left carotid, of the forceful impulses transmitted in a direct line from the left ventricle. In addition, these vessels of the circle are relatively of very small volume when one considers the overlying 1300 gm. brain bulk which pulsates strongly with the actual force of its blood supply carried to it by its comparatively delicate arrangement.

It would then seem that these four peculiarities: lack of surrounding structural support, tenuity of the vessel wall, the angulation of flow and relatively small calibre, might particularly predispose to dilatation and aneurysmal formation. Therefore it is suggested that these mechanical factors of stress and strain are sufficient producing causes and in the consideration of the presumptive etiology of these aneurysms, the factor of stress and strain peculiar to this location should be added equally important to the obvious arteriosclerotic degeneration and the possible congenital malformation. Such aneurysms might be called acquired mechanical defects. Any differentiation between a congenital and such an acquired origin is of course impossible but it is strongly doubted that they are all congenital.

The mycotic or inflammatory aneurysm is often spoken of and in the older literature was commonly reported as an autopsy finding. Today it is rearely seen clinically and is usually a terminal accompaniment of an acute endocarditis with sepsis. Ponfick[5] states that such an aneurysm can form and go on to rupture in nine days. No instance of the mycotic aneurysm was encountered in the author's series.

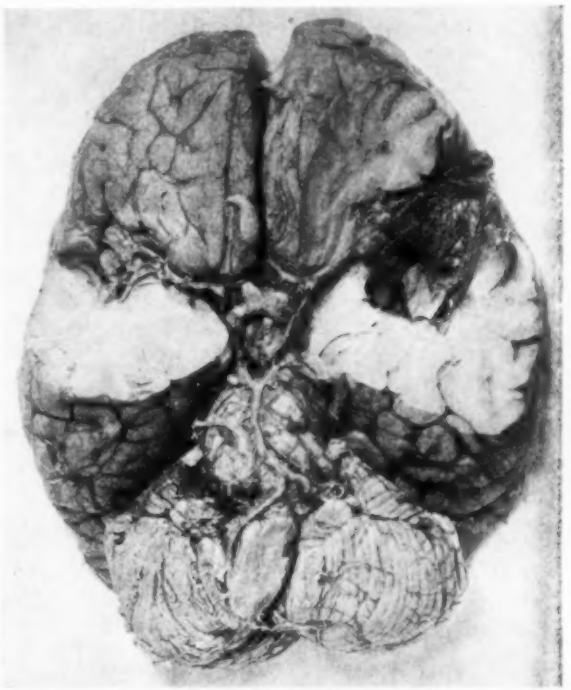

Fig. 2. Aneurysm on left middle cerebral artery in thirty-six year old woman who was seen in 1925 with definite spontaneous subarachnoid hemorrhage. She made an excellent recovery. Repeat attack in 1928 was fatal.

Collier[6] has suggested the possibility of a modified mycotic causation, theorizing that at some previous date an infectious disease might have caused microscopical intimal damage with a resulting weak spot in the vessel wall. At first glance this seems logical, but why then the frequent occurrence of aneurysms in the circle of Willis? According to Turnbull, cerebral aneurysms are much more common than those of peripheral vessels, occurring in the proportion of nine to one, if we exclude the specific aortic aneurysm. This type of inflammatory aneurysms should then be multiple but it is very unusual to find more than one. Syphilis is a surprisingly rare etiological factor. The possibility that previous cerebral trauma may cause the formation of an aneurysm has frequently

been mentioned in the older literature. Turnbull was unable to find any unequivocal evidence of such an aneurysm. It has not been favored but it is an interesting theory and will be discussed later.

We will then proceed with the premise that spontaneous subarachnoid hemorrhage is due to the rupture of a small aneurysm. The location and comparative size of these aneurysms described by the author is shown in the diagram (Fig. 3).

## SYMPTOMS

The symptoms of such a rupture depend upon the size and severity of the extravasation. In many cases there may be simple leaking for an indefinite period, with continuous or alternate oozing for several days and then recovery. Or there may be the large, spurting, tearing extravasation from a complete rupture which causes an apoplectic death in a very short time. As in cerebral apoplexy, death is rarely instantaneous, the shortest period being noted in the writer's series at thirty-five minutes. Because of the many cases reported at autopsy it might seem that it is a fatal disease. It is difficult to estimate the actual mortality as many patients recover without diagnosis, but from the writer's experience its mortality is not greater than 50 per cent.

The onset of this condition is precipitous, with excruciating, unbearable head pain, usually referred to the back of the neck. Some patients speak of a tearing, surging feeling. This is accompanied by marked vertigo and vomiting, and is usually followed by a variable degree of collapse. There may be in the severe cases complete coma and death within the first twenty-four hours. In the case of moderate severity the headache is intense, 20 of the author's series being seen for possible meningitis. There may be great dullness, but usually the patient is fairly well oriented. Pulse is slow; blood pressure may reach to surprisingly high levels—200+, erroneously suggesting a preexisting hypertension, but actually indicating the increased intracranial pressure. Eyegrounds may show a moderate degree of choking and many flame-shaped or diffuse hemorrhages.

It is to be remembered that the circle is in close approximation to the cranial nerves and involvement of a third or a sixth nerve is frequently seen. Occasionally there may be some laceration with softening into the brain stem, but rarely are there localizing signs and the case with a complete hemiplegia is best considered as intracranial hemorrhage or ordinary apoplexy. Fever as a reaction to the blood elements is always present. Occasionally a slight icterus may mislead to a consideration of biliary tract disease, especially if vomiting is pronounced.

The outstanding sign is the rigid neck; this is of the stone-wall variety and leads often to the preliminary diagnosis of meningitis. Spinal fluid tells the story. It is under increased pressure; it is uniformly bloody and, on centrifugalization, the supernatant fluid shows the characteristic amber hue of xanthochromia. This last occurs only after the first twenty-four hours. The sediment is bulk blood cells and it is surprising how small a sediment (0.1 c.c. bulk, to 15 c.c. fluid,) may produce a very hemorrhagic fluid.

## TREATMENT

With the initial puncture, the patient may obtain striking relief. This is not always true of subsequent taps and the argument waxes strongly as to therapeutic repetition of punctures; there are those that advocate frequent punctures, such as twice daily, while others, considering the danger of dislodging a sealing clot and inviting new bleeding, advise strongly against them. It is now the belief of the writer that the value of punctures has been overestimated. Many recover with one puncture only. Other presumptive cases undiagnosed in the early stage had no puncture and it would seem recovered just as rapidly as those with repeated taps. Histories strongly suggestive of spontane-

ous subarachnoid hemorrhage with recovery are frequently related.

A lumbar puncture has today become a very popular procedure. It is especially so in traumatic cases. There it undoubtedly has some virtue, but the writer fails to see such great value when it is routinely and indiscriminately performed two or three times daily just because the fluid is still bloody or there are a few persisting millimeters of increased pressure seen on a manometer, and it is believed that most patients recover in spite of it. It is now his plan to avoid, if possible, the initial puncture in the first twenty-four hours and then after the initial confirmatory tap to repeat it as infrequently as symptoms of increased pressure and curiosity may allow; its repetition may be avoided entirely in a great many cases. Nature does a beautiful work of absorption in the subarachnoid space and, judging by the surprisingly uncommon sequelae, the danger of the present-day much feared chronic arachnoiditis is not a great one. None of the recovered patients has shown the persistent headache, the insanity or epilepsy which are so often anticipated as sequelae from the chronic block arachnoiditis of traumatic subarachnoid bleeding. Such sequelae often flourish best in the low courts and may be better avoided by satisfactory financial settlement than by the repeated lumbar puncture. Early venesection when blood pressure is high might well be performed.

The two objects of treatment must be to relieve intracranial pressure and to promote sealing and healing of a bleeding stoma. The latter demands the elimination of all disturbing agitation in the vessel and in the tissue neighborhood, and again one is fearful of starting bleeding anew with the withdrawal of fluid. The patient must not be disturbed. Such masterly inactivity is often difficult of accomplishment. The people want action, but such action may be dangerous as when the duty-bound interne does his puncture on the sometimes resistant and unfortunate patient. Fluid should be removed slowly with an approximate reduction to half of the original pressure. Squirting fluid should be quickly checked with the stilette. Several days later the puncture for relief of the additional meningeal reaction to the blood elements might be less dangerous, but who can say when the vessel has stopped leaking? Eight of the fatal cases show repeat episodes of fresh bleeding before a final termination. Morphine has the greatest value and in the writer's opinion there is no objection to it. Elevation of the head may reduce some of the pumping power in the vessels. The intravenous glucose has no indication. It will reduce temporarily increased intracranial pressure by shrinking the brain bulk, but by so doing it could readily allow for more bleeding. Certainly intracranial pressure must have some virtue in damming the tide! However, decision is difficult and argument futile, only from the writer's experience he now seriously questions the value of too fre-

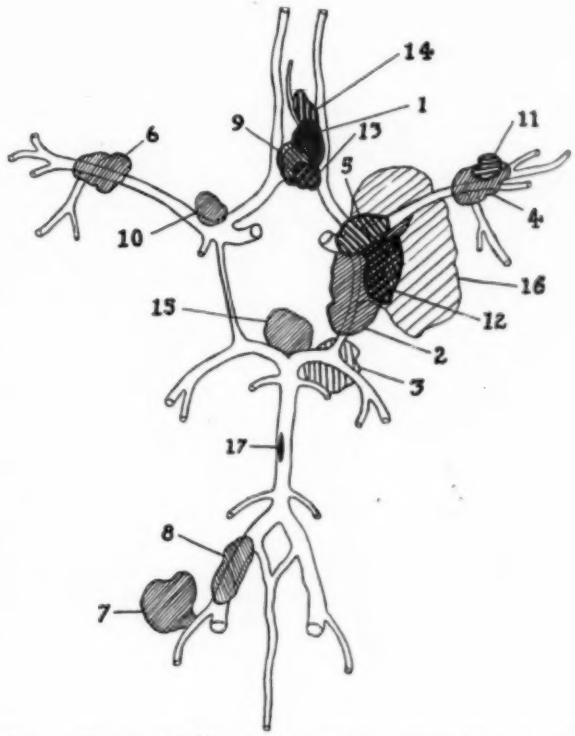


Fig. 3. Circle of Willis with position and relative size of 17 aneurysms.

quent lumbar punctures and strongly advises against them.

Can surgery offer anything? The possibility of clipping off a small berry aneurysm on one of the communicating branches might be considered, but it would be a most formidable procedure and exceedingly dangerous, and the writer fears quite impracticable. Injection of serum or thromboplastic substances to promote clotting is advised against.

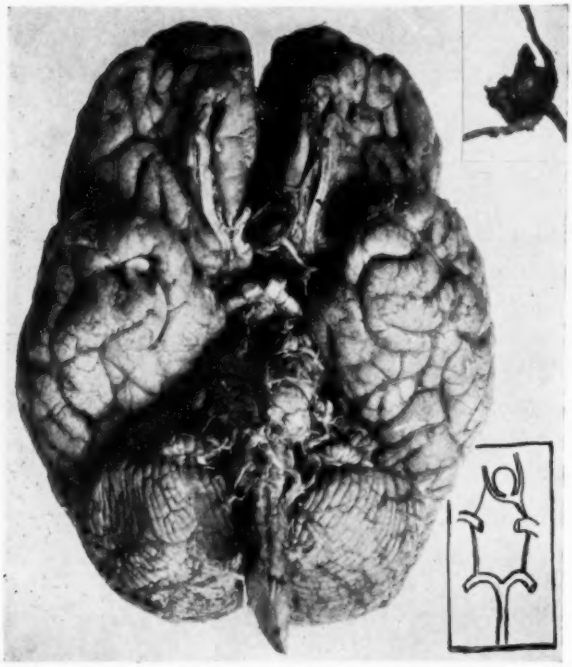

Fig. 4. Congenital aneurysm of anomalous left anterior cerebral artery.

The usual period of active illness for the recovered case is three weeks, but nine weeks arbitrarily should be set before any resumption of normal activities. They may "leak" again. Although a sacculated aneurysm, especially one with a narrow pedicle, may rupture, thrombose and heal completely, and this of course is to be hoped for, later ruptures may occur and if possible they are to be avoided. Such patients should eliminate sudden extremes of exertion and the onset so often occurs with some unusual strain. Eight of the entire series had fatal repeat episodes with intervals varying from two weeks to five years; one such case is shown in Figure 3.

As an illustration of the possible absorption of an aneurysm, the writer in 1912 saw his first case of spontaneous subarachnoid hemorrhage which was then diagnosed "hemorrhagic meningitis."

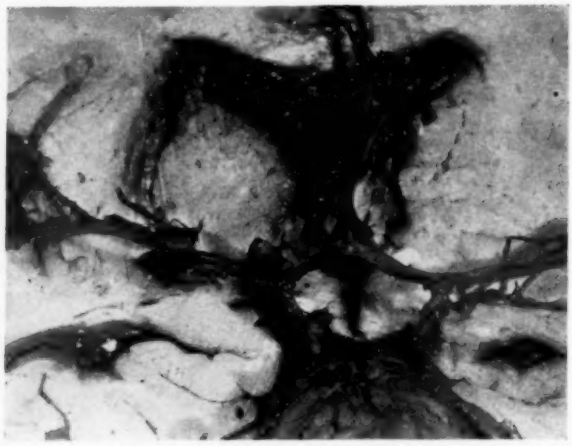

Fig. 5. Small aneurysm of right middle cerebral artery in twenty-year-old bell-hop who had, four years previously, suffered severe head injury in diving.

The patient was a forty-seven year old butcher previously well, without arteriosclerosis or recent injury. He made an excellent recovery and was followed closely during the ensuing years. There were no recurrences and he died in 1932 of gastric carcinoma. At autopsy there was no evidence of previous aneurysm found and careful dissection and study of the circle vessels and the surrounding brain tissue showed neither scarring nor evidence of aneurysmal formation.

It may seem unwarranted speculation but it is maintained that an aneurysm did exist originally.

Another case (Fig. 4) is a most unusual and excellent demonstration of healing of an aneurysm.

An Italian laborer of fifty years had five weeks of fever of undetermined origin; apparently there was severe headache, but the patient spoke no English and the history was inadequate. He recovered and two days after his discharge from the hospital he, with several others, was found dead in a small room from carbon monoxide poisoning. Postmortem clearly showed the nature of his previous undetermined illness in that there was a healing partially thrombosed aneurysm of an

anomalous anterior cerebral artery. The brain tissues around this region showed marked yellow discoloration.

Fig. 6. Sacculated, pedunculated aneurysm of right posterior inferior cerebellar artery. This aneurysm is displaced from its original position, tucked in between the cerebellum and medulla. The patient survived twenty-one days with three distinct episodes of fresh bleeding.

It is interesting to speculate what this man's future might have been. Twenty-four cases are still living, one having survived a second attack.

### MEDICOLEGAL ASPECTS

From the medicolegal standpoint the question of previous cerebral trauma as a cause of aneurysmal formation is of great interest.

A twenty-year old boy (Fig. 5), dying in six hours from the onset showed a ruptured small 0.5 cm. aneurysm on the left middle cerebral artery. Four years before he had a severe diving injury characterized by unconsciousness and three convulsions in two months following. Was the cerebral trauma the cause of the aneurysm?

A fifty-four year old woman (Fig. 6) who ten years before was injured in an automobile accident with severe wrenching and contusion of the neck and occipital region died of a ruptured aneurysm on the right posterior inferior cerebellar artery near its union with the vertebral.

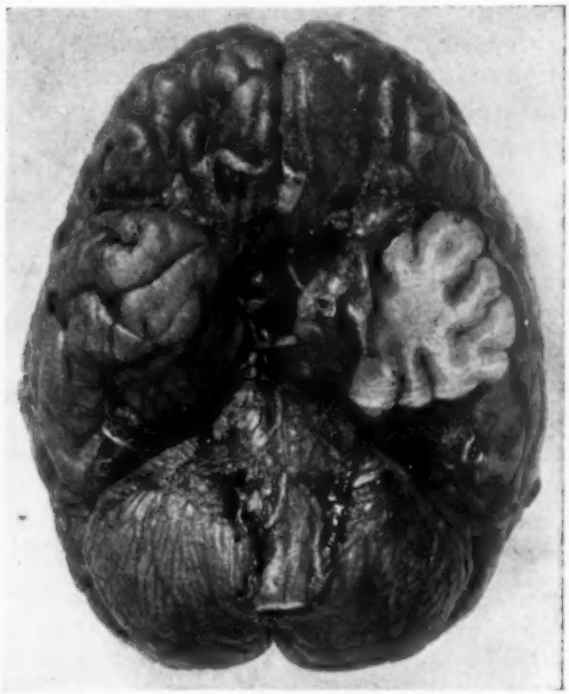

Fig. 7. Large aneurysm of left internal carotid in fifteen-year-old boy of normal development and mentality. At one and one-half years of age he suffered severe head injury. Subsequently complained of severe headaches and had two convulsive seizures. He died suddenly after two hours of severe headache, vomiting and terminal convulsion. Coroner's case.

A fifteen-year old boy (Fig. 7), a coroner's case, died suddenly of a large ruptured 4 cm. aneurysm. At one and a half years he sustained a severe head injury from a fall down a flight of stairs. Two recovered cases gave clear histories of severe head injuries, one with fracture, four and twenty years previously.

Is it possible that such previous, although remote, injuries may have caused minute vessel lacerations, leading to weakening of the vessel wall and subsequent dilatation with later fatal rupture? Such a theory may seem far-fetched and quite reminiscent of the traumatic causation of neoplasms, yet the latter, although not acceptable generally, has virtue in the minds of some when applied to cerebral neoplasms.

It seems reasonable that spontaneous subarachnoid hemorrhage from rupture of an aneurysm in an individual who had long previously sustained a severe cranial injury might well be the result of the original injury. It would be a difficult matter for absolute proof, but in the 5 cases cited it is felt that such remote trauma was a factor.

The causal relationship of exertion immediately preceding the attack is of considerable importance. In 18 of the author's series the onset was preceded by rather severe strain or possibly other significant activities. In one case, a thirty-five-year old woman was tusseling with her young nephew. A twenty-eight-year old woman was wildly cheering her player-husband in an exciting basketball game. Two attacks occurred while patients were working in extreme heat, one a painter on a roof. A twenty-four-year old woman was splitting wood. A thirty-eight-year old obese woman was struggling to get into a tight dress. Several cases occurred while straining at stool. Three occurred after severe osteopathic or chiropractic manipulations. In none of the author's personal cases did an actual medicolegal angle arise, but it has arisen with others in compensation practice. Similar contentions are frequent in regard to apoplexy. The problem is a difficult one, but it must be admitted that proved active straining-exertion would raise blood pressure and could well predispose to the rupture of an aneurysm, hence in accordance with the practice of most compensation courts in regard to aggravation it would be considered a sufficient producing cause and would be ruled compensable. Possibly it might seem unfair to charge such disability or fatalities against industry, because an aneurysm is at all times a potential danger and in all probability would rupture at some later time, possibly with the tying of a shoelace or even without exertion. There are rarely any signs that could suggest previously the existence of an aneurysm. The patient that develops apoplexy has had preexisting hypertension with some subjective evidence of headache and vertigo. He should be taken out of laborious activities, but there is no indication in the non-arteriosclerotic aneurysm. Many patients with spontaneous subarachnoid hemorrhage have multiple attacks and any person having had one attack of course should not be returned to onerous activities. Bruits although frequently mentioned are rarely heard and seldom listened for, and again unless the aneurysm is a large one there is nothing whatsoever to suggest it before rupture.

It is therefore believed that a man developing spontaneous subarachnoid hemorrhage during a severe strain of lifting or other heavy work while in occupation should receive compensation. It would definitely have to be shown, however, that at the time of such onset he was actually engaged in straining exertion. To the author's knowledge no case of aneurysm attributed to previous head injury has come up for consideration, but also it would be his opinion that a man who while employed had had a previous fractured skull or a severe cerebral injury as shown by unconsciousness, pressure and focal signs with resulting disability therefrom and who later develops a cerebral aneurysm which then ruptures should be compensated for this later disability or his family for a fatality on the premise that the aneurysm was dependent upon the previous trauma.

From the standpoint of exact diagnosis, it is important before spontaneous subarachnoid hemorrhage is subscribed to that the immediately recent head injury be excluded. Clinical and spinal fluid findings are often similar, and it might be a matter of some embarrassment to have in such a case a line of fracture demonstrated at autopsy. Traumatic subdural hemorrhage or the subdural hematoma is rarely to be confused with spontaneous subarachnoid hemorrhage. There may be a yellow spinal fluid, but it is not hemorrhagic. Usually these conditions are later developments of known trauma. A ruptured aneurysm

may occasionally produce subdural extension as well as the usual diffuse subarachnoid extravasation.

## CONCLUSIONS

1. Spontaneous subarachnoid hemorrhage is usually the result of a rupture in an aneurysm of the circle of Willis.

2. The qualification "spontaneous" is objected to as unsatisfactory and confusing.

3. The etiology of such aneurysms is difficult of determination. Aneurysms occurring without inflammatory or degenerative vascular changes usually have been considered congenital. It is suggested that many may be mechanical acquired dilatations.

4. The circle of Willis because of its peculiar arrangement and structure is subjected to unusual stress and may be in consequence susceptible to mechanical aneurysmal formation.

5. Previous severe cerebral trauma may be a factor in the formation of the cerebral aneurysm.

6. Exertion may, immediately preceding a rupture, be a cause of such rupture and hence has a medicolegal importance.

7. Approximately 50 per cent recover.

8. Aneurysms may partially rupture and heal to rupture again or they may thrombose and absorb completely.

9. Two per cent of sudden deaths are due to ruptured aneurysms.

Grateful acknowledgement is made to Drs. T. C. Wyatt and J. H. Ferguson for their assistance in the autopsy material, and to Dr. A. A. Getman for the privilege of including Case IV.

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# HEAD INJURIES

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IN automobile accidents alone in 1920, 11,061 persons were killed in the United States. By 1925 the number had increased to 19,610; in 1930 to 30,288; in 1933 to 33,500. In the city of Seattle with a metropolitan population of 550,000 the record for the past ten years shows a gradual increase in the number of deaths by automobile accidents from 48 in 1924 to an estimate of 97 (there were 57 during the first seven months) in 1934, giving us an increase of 100 per cent!

During the past four years the number of persons injured by automobiles alone in Seattle were as follows: 1931, 2863; 1932, 2987; 1933, 2765; 1934, an estimate of 2400, there being 1395 during the first seven months. More stringent enforcement of traffic laws, better traffic rules, and education of both the driving and walking public have helped in lowering the number of accidents, but the death rate has increased with alarming rapidity. Figures based upon the records in Harborview, King County Hospital, reveal a most startling story. In 1933, 1387 patients were admitted on account of injuries of such severity as to require hospitalization. Among these were 676 head injuries of various degrees. According to the Travelers Insurance Company 850,700 non-fatal injuries occurred due to transportation in 1933. In automobile accidents only one person in every 35 dies. Since a fairly large percentage of owners of automobiles carry some kind of liability insurance, or are financially responsible for damages resulting from an accident, a large number of these injury cases become medicolegal, and the record of each becomes part of the court record. Obviously a discussion of these cases might be valuable at this time.

A cerebrocranial injury is one in which both the brain and skull are involved. "Cerebral trauma," "concussion of the brain," "contusion of the brain," "skull fracture," "laceration of the scalp," and "compound skull fracture" are all included in the general term "head injury." From the legal point it is better to be more general in terms than specific when making the first reports. Later specific terms are essential. For purposes of this article the general term "head injury" will be employed first; when discussing the legal status of post-traumatic sequelae specific definitions and terms will be used.

Twenty years ago anyone receiving a severe head injury would be taken to a hospital and given such treatment as the individual surgeon who had charge of the case would prescribe. None of us knew very much about the rationale of treatment. From a medicolegal standpoint those injuries were not particularly important, and comparatively rare. The subsequent results, both functional and organic, had not been given a definite legal status at that time. Twenty years has seen a great change in the medical care of a severe injury to the head involving either the skull, brain or both. The medicolegal aspect has developed and has caused the medical profession as a whole to familiarize itself with many factors formerly considered unnecessary. We must include the entire medical profession when we discuss head injuries since many are treated by the country practitioner, the surgeon in smaller communities, the specialist in larger centers, and finally the highly developed group organizations in the modern hospitals of today. The legal fraternity has found these cases to be a rather lucrative part of their practice. Certain men in that profession

are really specialists in this type of work. It, therefore, has developed that today the physician owes to his patient a certain responsibility of a medicolegal nature outside of medical and surgical care.

Immediately upon assuming charge of an accident case involving the head, certain routine procedures should be performed. A brief but accurate description of the accident should be obtained. At the same time it is not difficult to get some of the essential details, that is to say, whether the patient was driving the car in the case of an auto accident, or what he was doing in any case; those with him and what happened to them, and many other incidental facts which may be of import at a later date. The old army dictum "when, where and how" is quite applicable to the civilian accident. It is always important to take names and addresses of people, related or unrelated, who are close to the patient. Owing to the confusion associated with accidents these details may be overlooked unless it is done as a matter of routine.

Having established these facts for record, the examination of the patient is the next step, and a record of this examination is imperative. It is of little benefit to either the patient or the hospital if several months after the patient has left the hospital, the court requests hospital records only to find little or no evidence of the examination. *In court one cannot testify to what he thinks was the condition of the patient. The record must show what actually was found to be present upon examination.* It is well also to make a notation of those present at the time of the examination, such as doctors, nurses, relatives or friends. It is not essential, perhaps, but well enough to have this information for any future reference. We can take the advice of the great surgeon, Murphy, who instructed his internes to "let the record show," as well as that of another great Irishman who said "Let's have a look at the record." Finally, it is the record that the jury reads.

Today most of the hospitals in the larger centers are fully equipped and staffed to handle emergency cases. Accidents, however, do not always occur at the site of a standardized hospital. Frequently the most serious accidents occur in the country districts, and the patients are taken to small suburban hospitals that are not sufficiently equipped and staffed to handle serious accident cases involving the head. In such institutions serious consequences may easily be avoided by judicious use of the telephone and ambulance.

Every neurosurgeon knows that in severe head injury cases the first twenty-four hours mean either a fatal termination or a chance to fight it out with some hope of winning against death. He also knows that today there are only a few essentials to the efficient care of these cases for the precious first twenty-four hours. Therefore, any hospital, in fact almost any home with the physician's first-aid kit, can have at hand these essentials, the appliances being (1) sterile gauze, (2) bandages, (3) a bed, (4) sphygmomanometer, (5) chart, (6) large syringe for intravenous medication; and the medicines, (1) atropine (hypodermic), (2) camphorated oil or other mild stimulant, (3) magnesium sulphate, (4) sodium sulphate, (5) chloral hydrate, (6) morphia sulphate, (7) adrenalin, (8) glucose ampules, and (9) antiseptic solution. This is not at all a formidable array either of appliances or medical equipment. A spinal manometer, restraint or straight jacket and blood transfusion apparatus are only occasionally required during this twenty-four hour period. *X*-rays and complete neurological examination only add to the shock. Both may be well reserved for a later period when they can be made more easily and not harm the patient materially. After the period of shock has passed, which is usually within twenty-four hours, the patient can be taken to a larger hospital.

One important factor in the proper management of any head injury case is the choice of a nurse or nurses. No other traumatic case is so at the mercy of outside influences as the one suffering from a

severe head injury. With returning consciousness suggestions from all sources are deeply imbedded in the receptive mind of the patient. It is important, therefore, that the ones in closest contact with the patient be both intelligent and sympathetic. To this end the nurses should understand the psychology underlying the recovery of head injury cases. They should be acquainted with the materials which the physician will use for the physical recovery and in a similar way should be acquainted with the factors which will tend to assist in mental recovery, and to avoid such situations as might retard it. This is not always easy, but from both medical and legal angles may play an important part.

The question of visitors, relatives or friends, must be given consideration from the very beginning. They should be kept informed as to progress from time to time, and should be permitted to see the patient as soon as he has recovered from shock. However, their stay in the room should be of very short duration. Frequently attorneys stress the fact that the patient was so ill that neither relatives nor friends were permitted to enter the sick room. This may be misleading to a jury. On the other hand to have visitors about is sometimes more harmful than beneficial to the patient. Above all things the suggestion of future disability must be kept from him. This at times is difficult, but is absolutely imperative.

Post-traumatic hysteria always begins as a suggestion from some one to the effect that the patient has had a serious injury from which he may not fully recover. *This is always the beginning of any case of post-traumatic hysteria.*

For all of these reasons it is essential a nurse be well trained, intelligent and watchful of the patient's interest. At the same time she should be most careful that she herself does not cause trouble during this period of susceptibility. This applies to both male and female patients. Women crave sympathy to a greater extent than men, and normally are more emotional and respond more easily to external influences. Under such conditions it is so easy to offer suggestions, and only the physician and neurologist know how thoroughly they are retained.

I once said that "nurses on head injury cases should be beautiful but dumb," beautiful in their care of the patient, dumb about all matters pertaining to the accident and its after effects. Such a nurse is not hard to find, and hard to keep.

## SPINAL PUNCTURES

An important medicolegal factor has to do with the question of spinal punctures. A smart lawyer will make use of the facts either for or against the patient's interest depending upon whether a spinal puncture was made or not.

In a questionnaire undertaken by Dr. Harry Mock of Chicago relative to the custom of various neurosurgeons on the use of spinal punctures in head injury cases he writes as follows:

> In a general way, it will be of interest to you to know the results of the questionnaire:
> Dr. Sachs and Dr. Dandy are opposed to lumbar punctures in acute craniocerebral injuries.
> Dr. Cushing and Dr. Swift favor routine lumbar punctures in all such cases.
> Drs. Bailey, Davis, Fay, Frazier, Naffziger and Peet favor the procedure in selected cases—some of these men use it almost routinely, while the others use it less frequently.
>
> My position has been to use lumbar puncture in those patients showing certain signs and symptoms which, if allowed to continue, would most probably go on into medullary compression. Thus, approximately 30 per cent of my cases have had lumbar puncture.

Unless the patient has had bleeding from the ears, cerebrospinal fluid escaping from nose or ears, or very definite *x*-ray findings, one cannot say with certainty (legally) that the patient has suffered either injury to the brain or meninges in many cases. On the other hand, a spinal puncture with withdrawal of only a very few drops of

fluid, a manometric reading and a record of the findings not only does *no* harm to the patient but protects him legally, should positive findings be obtained.

Spinal drainage is another matter. Some neurosurgeons use it in preference to hypertonic solutions routinely to prevent and to relieve cerebral edema. My own procedure is to advise it when in consultation whenever I am convinced that edema is either threatening or present and hypertonic solutions may not be used with discretion. When to use glucose solution intravenously and when magnesium sulphate per rectum is so difficult to determine for the average physician that frequently one is much safer in the use of spinal drainage. They are both powerful remedies and should be used with caution. Certainly an accurate record of the pulse and blood pressure should be kept so that, if the question arises, the record justifies the use of either or both. One should never use either unless the pressure signs positively indicate interference along these lines.

### X-RAY FILMS

It is quite unnecessary to mention the accurate identification of *x*-ray films. Both positive or negative findings are important to one or the other side of a case. Certainly the facts as shown by the film should be placed on record. This cannot be done unless they are properly marked. The hospital record also should have a written report from the radiologist. One not familiar with the lines on the *x*-ray film made by the various blood vessels and sutures should not presume to pass upon a film as to the presence or absence of a fracture.

### TERMINOLOGY

Potentially all injury cases arising in civil and industrial life may at some future date become medicolegal in character. We are interested only in those involving the cranial cavity and its contents. Unfortunately, severe injuries to the brain may frequently cause severe mental changes which complicate the picture. Mental changes may be either of functional or organic nature, an additional factor to be considered.

Table I

NATURE OF NON-FATAL INJURIES SUFFERED BY AUTOMOBILE ACCIDENT VICTIMS IN 1932*

| | *Persons Injured* | *Per Cent* |
|---|---|---|
| Fractured skull | 10,770 | 1.19 |
| Fractured spine | 1,540 | .17 |
| Other fractures | 67,680 | 7.48 |
| Concussion of brain | 10,770 | 1.19 |
| Severe general shock with contusions and lacerations | 207,920 | 22.98 |
| Shock (less severe) and shakeup | 548,310 | 60.60 |
| Internal injuries | 11,760 | 1.30 |
| Other injuries (sprains, dislocations, wrenches, etc.) | 46,050 | 5.09 |
| Total | 904,800 | 100.00 |

Table II

NATURE OF NON-FATAL INJURIES SUFFERED BY AUTOMOBILE ACCIDENT VICTIMS IN 1933*

| | *Persons Injured* | *Per Cent* |
|---|---|---|
| Fractured skull | 11,830 | 1.39 |
| Fractured spine | 1,700 | .20 |
| Other fractures | 68,140 | 8.01 |
| Concussion of brain | 12,500 | 1.47 |
| Severe general shock with contusions and lacerations | 196,340 | 23.08 |
| Shock (less severe) and shakeup | 489,830 | 57.58 |
| Internal injuries | 11,490 | 1.35 |
| Other injuries (sprains, dislocations, wrenches, etc.) | 58,870 | 6.92 |
| Total | 850,700 | 100.00 |

* Tables I and II compiled by the Travelers Insurance Company of America. Approximately 3 per cent of all injuries in the years 1932 and 1933 involved the brain, skull or spine. If one is to include "severe general shock" the percentage is approximately 25.

Legal terminology is quite different from medical terminology so that our problem becomes more complex. A person may be insane legally, and yet perfectly sane medically. Insanity is a legal term and must be considered as such. Neurosis, psychosis, hysteria, malingering, etc., are

terms having a somewhat different meaning when bandied about in the court room in the presence of a jury.

Terms that are used to describe certain conditions as results of trauma should be used with some degree of intelligence. It is doing one's patient a great injustice to write down on the record that he is suffering from a traumatic psychosis, for instance, when as a matter of fact he has developed a post-traumatic psychosis. Hysteria is one of the most common terms to be used in describing the sequelae following trauma. No effort is made to differentiate between hysteria, malingering or a true psychosis. However, when the case comes before the jury, an alert lawyer will question the jury relative to the terms, asking his questions in such a manner that the jury will be confused, and the testimony, to a certain extent, discredited.

Terms to describe the mental symptoms are also used more or less indiscriminately by those not well versed in psychiatric terminology. A traumatic psychosis usually indicates more or less confusion, delirium and disorientation occurring at the time of the trauma with no intervening period between the trauma and the mental illness unless it be unconsciousness. Post-traumatic psychosis is used to denote a severe mental illness which had developed because of severe brain damage of a progressive nature and beginning, usually, a considerable time after the trauma. It generally requires hospitalization. According to Wechsler traumatic hysteria denotes the occurrence of symptoms of hysteria which follow an injury wherein the individual unconsciously makes use of the accident to solve personal difficulties which have no relation to the trauma. Traumatic neurosis and post-traumatic neurosis are commonly used interchangeably, post-traumatic neurosis being the better term. This denotes usually the host of symptoms which often occur after a so-called "incubation period" such as the elaboration and increase of headache, dizziness, irritability, memory loss, inability to concentrate, etc., and whose descriptions are highly suggestive of symptoms which might arise from organic change.

Malingering is the conscious and voluntary effort to feign an illness which an individual knows is not present for the purpose of some gain for himself.

Neurasthenia is that symptom complex "characterized by lack of energy, fatiguability, mild gastrointestinal disturbances, various pressures on the head and spine, etc., usually associated with prolonged or excessive masturbation" (Wechsler). While this definition may be disputed, neurasthenia should never be used in connection with trauma as an etiological factor.

Sanity and insanity are likewise terms that are frequently improperly used in dealing with post-traumatic conditions. The patient may be suffering from a traumatic psychosis in which case he is not legally sane. That means that legally he is unable to take care of himself either from a physical standpoint or mental standpoint, or both. Insanity, therefore, is a legal term and not a medical one. To state that a patient has become insane as a result of a head injury means this patient has become unable to meet the requirements of the law insofar as his actions are concerned, but does not have any descriptive meaning from the medical standpoint.

## RELATIVE IMPORTANCE OF HEAD INJURIES IN COMPARISON WITH OTHERS

Statistics are difficult to obtain in this respect. Probably one of the most comprehensive tabulations compiled from year to year is published by the Traveler's Insurance Company of Hartford. Tables showing the number of head injuries, concussion of the brain, and skull fracture is shown in Tables III and IV. Approximately 3 per cent of all injuries are classified as head injuries. These do not include "general shock cases." If it is to be included, and it is generally cerebral in origin, the percentage is 25. In a careful survey of all admissions to Harborview, King County Charitable Hospital of 600 beds, perhaps a more

accurate conclusion can be made. All case records were checked and the figures are shown in Figure 1. From this it appears that nearly 50 per cent of all injuries caus-

the influence of the automobile in a city of hills and slippery streets during the rainy season.

With such figures before us showing

Table III

TYPES OF ACCIDENTS RESULTING IN PERSONS KILLED AND INJURED IN 1932*

| | *Number of Accidents* | *Per Cent* | *Persons Killed* | *Per Cent* | *Persons Injured* | *Per Cent* |
|---|---|---|---|---|---|---|
| Collision with: | | | | | | |
| Pedestrian | 291,190 | 39.07 | 12,770 | 44.0 | 267,280 | 29.54 |
| Automobile | 323,830 | 43.45 | 7,160 | 24.7 | 471,850 | 52.15 |
| Horse-drawn vehicle | 4,920 | .66 | 100 | .4 | 5,790 | .64 |
| Railroad train | 4,020 | .54 | 1,140 | 3.9 | 4,710 | .52 |
| Street car | 7,970 | 1.07 | 260 | .9 | 10,590 | 1.17 |
| Other vehicle | 9,910 | 1.33 | 340 | 1.2 | 10,590 | 1.17 |
| Fixed object | 42,710 | 5.73 | 3,110 | 10.7 | 57,180 | 6.32 |
| Bicycle | 15,580 | 2.09 | 400 | 1.4 | 15,110 | 1.67 |
| Non-collision | 42,560 | 5.71 | 3,600 | 12.4 | 59,080 | 6.53 |
| Miscellaneous | 2,610 | .35 | 120 | .4 | 2,620 | .29 |
| Total | 745,300 | 100.00 | 29,000 | 100.0 | 904,800 | 100.00 |

Table IV

TYPES OF ACCIDENTS RESULTING IN PERSONS KILLED AND INJURED IN 1933*

| | *Number of Accidents* | *Per Cent* | *Persons Killed* | *Per Cent* | *Persons Injured* | *Per Cent* |
|---|---|---|---|---|---|---|
| Collision with: | | | | | | |
| Pedestrian | 280,960 | 37.14 | 13,440 | 44.95 | 262,270 | 30.83 |
| Automobile | 339,440 | 44.87 | 7,150 | 23.91 | 434,110 | 51.03 |
| Horse-drawn vehicle | 5,750 | .76 | 200 | .67 | 5,610 | .66 |
| Railroad train | 4,310 | .57 | 1,360 | 4.55 | 4,430 | .52 |
| Street car | 10,820 | 1.43 | 260 | .87 | 10,800 | 1.27 |
| Other vehicle | 7,870 | 1.04 | 300 | 1.00 | 8,420 | .99 |
| Fixed object | 46,370 | 6.13 | 3,140 | 10.50 | 56,740 | 6.67 |
| Bicycle | 16,720 | 2.21 | 420 | 1.41 | 15,910 | 1.87 |
| Non-collision | 41,910 | 5.54 | 3,570 | 11.94 | 50,450 | 5.93 |
| Miscellaneous | 2,350 | .31 | 60 | .20 | 1,960 | .23 |
| Total | 756,500 | 100.00 | 29,900 | 100.00 | 850,700 | 100.00 |

* From Tables III and IV compiled by The Travelers Insurance Company of America it is seen that there were approximately 750,000 accidents. In 1933 there were 850,700 persons injured, while in 1932 there were 904,800. Since approximately 3 per cent were head injuries and subject to post-traumatic sequelae, it is quite obvious that this class of patients should have the most careful attention. The post-traumatic sequelae in the majority of cases are results of suggestions to the patient from outside sources.

ing hospitalization of a patient involve the head.

The curve for the entire year of 1933 shows the number in the summer months to be less, but there is an increase in August and the peak is reached in October. This is of interest since it demonstrated conclusively that head injuries constitute 50 per cent of the total of severe injuries in large centers, and 25 per cent in a nation-wide tabulation which would include all means of transportation, it is quite obvious that this group of cases must offer the greatest opportunity for

medicolegal controversy. Let us look at the report furnished by the New York Life Insurance Company.

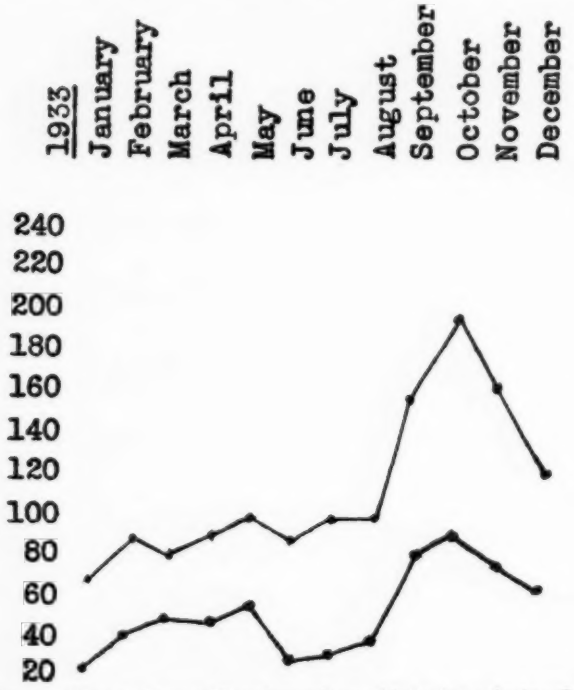


FIG. 1. *Upper Curve:* Total number of injuries admitted to Harborview, King County Hospital, during the year 1933. *Lower Curve:* Number of head injuries included in this group. The peak occurs in September, October and November, showing the influence of automobile traffic in Seattle.

During the year 1933 the New York Life had deaths among 16,182 policyholders, of whom 1,468 (9.1 per cent) were classed as accidents. Out of 720 accidental deaths under which double indemnity was paid, 333 (43.4 per cent) were classed as caused by automobiles. Thus it may be assumed that 3.9 per cent of all deaths were due to automobiles either as pedestrians or as passengers. Not all these automobile deaths will have entailed head injuries and there were other accidental classes which may have involved such injuries. Thus, for example, falls caused 51 deaths, homicide 51, gunshot 30, railroad 19, crushing accidents 14, and hunting 11 deaths. Insurance company statistics do not specify as a rule whether the head was injured or not. Likewise there is no mention of the location of the injury in the International List of Causes of Death, as used by the U. S. Census Bureau at Washington, D. C.

In a mortality study made by a large number of companies and published in 1931, there was shown for the first time a mortality experience on lives accepted for insurance with a history of fractured skull. Such cases were generally declined within one year after the fracture occurred; the cases with more remote histories were divided into two groups, according to whether they were accepted at standard premium rates or restricted to the payment of extra premiums on account of the history of fractured skull. There were 54 deaths in the first group and a standard mortality, while in the second group there were 22 deaths with twice the standard mortality.

This is conclusive proof that great organizations dependent entirely upon actuary statistics for the success or failure of their financial structure, consider head injury cases the most serious cases from the standpoint of re-insurance. Physicians the country over must become more conscientious in the detailed management of these cases. Their records should be kept with the greatest care so that the rights of their patients may be guarded.

# THE MANAGEMENT OF UROLOGIC COMPLICATIONS IN INJURIES TO THE SPINE

## REPORT OF 54 CASES WITHOUT A SINGLE INFECTION IN THE URINARY TRACT*

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ALTHOUGH the purpose of this article is to discuss a purely urologic problem, it is presented before a surgical group because the general surgeon is the one who first comes in contact with this type of case. It is to be regretted, however, that the contributions of general surgeons on this subject either omit to mention the occurrence of urologic complications or else do so very inadequately.[1–5] On the other hand, there is no unanimity of opinion amongst urologists as to how to treat these complications when they arise; the result is a discouragingly high mortality rate.

The statistical work of this paper is entirely due to my confrere, Dr. Nash; my part is only the clinical aspect, in which I have been deeply interested for many years. In 1914 we had 3 cases of spinal injury on our service at Harlem Hospital, two were gunshot wounds and the third a compression fracture. All three patients died as a result of urinary infection. We decided at that time that the infection was caused by catheterization, and ordered that no case of spinal injury was to be instrumented. But like many other efforts of this kind, our advice and suggestion was not taken by our fellow-workers. Only last fall I saw a private case of spinal fracture; this patient was catheterized as soon as his bladder became distended; the result was fatal, as was to be expected.

### STATISTICAL DATA

Injuries to the spine occur very frequently in both military and civil practice. While the two types of cases are not to be confused, a great many lessons in the management of these cases have been learned from treating cases injured on the battlefield.

An incomplete summary issued by the U. S. Army[6] gives the number of spinal injuries as 2324 during 1917 and 1918. A large series of injuries to the spine are reported by Thompson Walker[7] for the British Army. Drapier,[21] Wertheimer,[28] Dossot,[29] etc. report on the experiences of the French Army. The writers addressed letters of inquiry to the German and Austrian War Offices, but were told that there were no statistics available.

In civil life a large series is reported by Sante and McCutcheon, (quoted by Christopher[20]) 400 cases; Menard, (quoted by Zesas[16]) 382 cases; Conwell,[15] 215 cases; Haumann (quoted by Thomas) 204 cases; Hartwell,[8] 133 cases; Thomas,[9] 100 cases.

These figures are very important when the mortalities resulting from the spinal injuries are considered. Very grave results appear. Thus, the U. S. Army[6,11] report states; "Fully 80 per cent of them die in the first few weeks in consequence of infection from bed sores and catheterization." The figures for the British Army[7] closely approximate those of the U. S. Army mortality figures.

Thomas,[9] whose cases were for the most part industrial casualties, reports a mortality of 36 per cent; Haumann, 50 per cent; Henry,[12] 81 per cent, and Wallenstein,[13] 80 per cent.

### PATHOLOGIC PHYSIOLOGY

It is generally agreed that the most important complication in injuries to the spine is involvement of the urinary tract.

* From the Surgical Service, Harlem Hospital. Read before the Section of Surgery, New York Academy of Medicine, May 4, 1934.

When the urologist is called to treat the urologic complications, he often makes the error of comparing the disease process here to other uropathies. Obstructive lesions at the bladder neck causing retention, such as adenoma of the prostate, median bar, etc. are in no manner to be compared to urinary retention following injuries to the spine. Even tabes with its specific pathology in the spinal cord must not be regarded in any sense as presenting an analogous problem.

The changes which occur in the spinal cord as well as in the urinary tract following an injury may now be reviewed briefly.

A. Changes in the Spinal Cord. The pathology which is to be found in any individual case will depend upon the manner in which the injury was caused, i.e. whether gunshot wound, industrial accident, etc.; the nature of the injury, i.e. fracture, dislocation; as well as the location of the injury, i.e. cervical, dorsal or lumbar region. According to Sharpe,[1] the symptoms of cord injury are due to the following:

"(1) Fragments of bone causing contusion and lacerations of the nerve fibers.

(2) Hemorrhage into the cord wounds.

(3) Edema within and around the cord."

If the case goes on to chronicity we may have:

(4) "Narrowing of the spinal canal by new bone growth or scar tissue formation."

When the cord substance is injured the following conditions may arise.[7]

(a) *Concussion of the Cord.* This is a mild condition, due to a subsiding edema for example, causing urinary dysfunction which may clear up in a few days.

(b) *Compression or Destruction of the Vesical Center.* Clinically, this is the type most frequently met with because injuries of the spine occur most commonly at the first and second lumbar vertebra. When this condition arises, the bladder is released from the influence of the cortical, subcortical and vesical centers. For a period of from several days to several weeks there is retention of urine, but sooner or later the bladder becomes susceptible to the sympathetic system through its connections with the hypogastric and inferior mesenteric ganglia, then the bladder evacuates itself at intervals. This is the so-called *automatic bladder.*

(c) *Destruction of the Cord above the Vesical Center.* In this type of injury there is a complete paralysis of the bladder and sphincter. The influence of the higher centers of the brain have been entirely removed, and stimulation of the "lower portion of the spinal cord may not bear the character of a local signature."[17] This phenomenon was studied by Mayo Robson,[18] Sir Francis Kidd,[17] Henry Head and George Riddoch,[19] etc. Normally, when the sole is tickled the stimulus will cause withdrawing of the foot, but if the local signature is absent, the response to such a "stimulus is in the nature of a mass reflex, that is a pouring out of motor energy to other structures than the foot. By means of this reflex the bladder may be made to empty."[17]

B. Changes in the Kidney. Zesas,[16] writing in 1910, points out that following an injury to the spine the kidneys may respond by a hyposecretion or a hypersecretion; he reports a proved case of suppression of urine in a man who died eight hours after the injury. He states that clinically oliguria is more frequent, but polyuria also occurs; glycosuria, albuminuria, and hematuria have also been observed. He believes that the cause of all these symptoms is a "vasomotor phenomenon or perhaps a specific secretory nerve lesion."

C. Changes in the Bladder. Normally the bladder is controlled by:

(a) Cortical and sub-cortical centers (thalamus, etc.)

(b) Vesical center in the spinal cord.

(c) Ganglia (hypogastric and inferior mesenteric).

As a result of injuries to the spine, the function of the bladder may be remarkably influenced as already noted. Either the higher or lower centers or both may be removed. In addition to that a notable

change in the function of the kidney may also occur as was just seen. The combined effect of all these phenomena is to produce an infection in the urinary tract. The precise causes for this infection may now be studied.

### ETIOLOGY OF URINARY INFECTION IN SPINAL INJURIES

*Predisposing Causes.* Infections in the urinary tract have been long recognized as the most important contributing cause of death in injuries to the spine. Dennis[14] pointed this out when he discussed the injuries in the Civil War, "Too much stress cannot be laid upon the great importance of preventing cystitis which in turn establishes a pyelitis and eventually leads to death." The factors contributing to this infection have been appreciated only recently however. Zesas believed that infection is due to "a trophic disturbance of the mucous membrane in the bladder similar to a decubitus ulcer on the body." He quoted Kocher and Alberran who coincided with this opinion, and added that residual urine in the bladder is a favorable culture medium for bacteria. This view is amplified by Drapier[21] who points out that infection is due to a disturbance of *innervation* of the bladder:

(a) There is a loss of *motor* innervation which produces poor emptying, stagnation of urine, fermentation, and therefore, a good culture medium for bacteria.

(b) There is a loss of *sensory* innervation of the mucous membrane of the bladder (and urethra), and these may then be injured very readily by instrumentation.

(c) There is a loss of *trophic* impulses of the mucous membrane, removing the physiologic power of defense and permitting more easy entrance for bacteria. To this trophic disturbance "there is added a tremendous vasodilatation. . . sometimes even a suffusion of blood into the submucosa or hemorrhage . . . excellent terrain for bacteria."

Some authors[22] have felt that changes to the nerve supply of the ureter bring about a paralytic relaxation of the ureteric sphincters, and thus cause infection in the upper urinary tract. This idea was proved to be erroneous by the very excellent work of Fullerton[23] who showed that the "peristalsis of the ureter continues."

*Source of Infection.* In 1917 F. A. Besley,[24] discussing war injuries of the spinal cord, wrote that he had never seen a case of cystitis in a patient who had not been catheterized. Two English surgeons, Vellacott and Webb-Johnson,[25] in an article published in 1919, support the view that infection is carried by instrumentation. They report a small series of 10 patients, none of whom was catheterized before admission; only one of these developed a very mild urinary infection due to B. coli. In a larger series of 40 patients all of whom had been catheterized before admission, 31 or 77 per cent developed urinary infection. Briefly stated, infection may occur without catheterization by bacteria reaching the bladder from:

(1) The digestive tract

(2) The urethra

(3) Excretion of organisms by the kidneys

(4) Absorption of organisms from septic wounds.

The last mentioned source is important in war injuries only. The first three undoubtedly account for incidental infection such as that described by Vellacott and Webb-Johnson, but play no part in the large mortality and morbidity statistics in the opinion of some of the best commentators on the subject (O'Connor, Hagner, Alcock, Townsend, Chute, Quimby, etc.[21]).

In contradiction to the overwhelming opinion just cited, one must quote from Sir Robert Jones[30] who states: "Cystitis occurs almost, if not quite as commonly, when no catheter has even been passed." A very careful search of the literature prompts the writers to conclude that Sir Robert Jones is the only one who made these observations. Also, 49 patients whose cases are reported in this article had not

been catheterized; not one of them developed any urinary infection.

*Organism.* The causative organisms in the order of their frequency of infection are:

(1) B. coli.
(2) Mixed B. coli and cocci.
(3) Streptococcus.
(4) Staphylococcus.
(5) B. pyocyaneus.

## TREATMENT

It seemed worthwhile to the authors to study the experiences of the warring nations in cases of injury to the spine, and upon inquiry the following information was obtained:

(1) U. S. Army:[6] "Just what should be done for the bladder in these spinal cases was never a matter for general orders. There remained a difference of opinion whether permanent drainage, intermittent catheterization, or abstention from any intervention, merely allowing the bladder to fill and overflow by dribbling was the method most likely to forestall infection."

(2) British Army: In general the treatment was the same as the United States Army.

(3) French Army: In the early part of the war the treatment was catheterization, but later there was a strong leaning to suprapubic cystotomy as a prophylactic measure to prevent infection.

(4) Germany Army: The literature does not contain any publication touching upon the experiences of the German medical officers. The authors wrote to the German war office, and were regretfully informed that there are no mortality or morbidity statistics, and nothing was mentioned about the method of treatment.

(5) Austrian Army: Here again there are no published data. Communication with the war office in Vienna elicited the same reply as from the war office in Berlin.

The first method of treatment which is usually recommended to relieve urinary retention is catheterization. Some authors distinguish between intermittent catheterization and treatment by indwelling catheter. In practice, there is no difference between the two methods; the first probably causes more trauma, the second is probably less effective in evacuating blood clots. Both methods invariably lead to infection.

The purpose of this article which is based upon the study of 54 cases is to prove that the avoidance of catheterization is absolutely essential in the management of urologic complications following injuries to the spine. It must be said at the outset that this idea is not new. Besley,[24] an American military surgeon, writing in 1917 condemned the use of the catheter in his "A Plea for the Non-catheterization of the Urinary Bladder." Hanson[10] writing in 1930 expressed the same opinion. A host of other writers: Vellacott and Webb-Johnson,[25] V. J. O'Conor,[26] Esaw,[27] Gurdjian,[7] Zesas,[16] etc., all condemned the use of the catheter. The latest article on the subject was one by Robert E. Cumming;[22] he states: "I believe that the greatest . . . danger . . . lies in this practice (catheterization) . . . " Unfortunately, however, the case reports show that every one of Cumming's patients had been treated by catheterization. If one may borrow a phrase from our friends, the politicians, urologists appear to be rendering lip service when they condemn the use of the catheter, but in practice they all use it.

The second method proposed for the care of urinary retention is suprapubic cystotomy. This was developed by the French Army, and is still recommended as the method of choice by Drapier[21] and Wertheimer.[28] It may be of historical interest to add that Dennis, in his description of the injured during the Civil War, writes, "The operation of perineal cystotomy has been performed . . . by Mr. Darrier Harrison who inserted a rubber tube into the bladder through the perineum, and attached to the tube a piece of India rubber tubing, which carried the urine from the bladder to a vessel upon the floor. The bladder was

washed out twice daily by antiseptic solutions."

A third method of treatment which has been suggested in the care of the bladder is "manual expression"[16] (suprapubic pressure[7] or Credeing[17]). The consensus of opinion is that when properly employed this method is very valuable because it provides a means of evacuating the bladder that does not entail the danger of infection.

In Harlem Hospital we have had an opportunity to study all of the three methods. Each method was tried, but the mortality statistics continued at the usual high level because at one time or another catheterization had also been employed. Finally we decided that the only way to test out the "let alone" policy was to really avoid catheterizing these patients.

Between 15 and 25 cases of spinal injuries are admitted to Harlem Hospital each year. The 54 cases concerned in this series constitute the admissions since 1929 when the rule was laid down that no patient whose bladder is distended as a result of a spinal injury is to be catheterized. The cooperation of both the internes and the attending staff was enlisted in order to carry out this rule. Nevertheless, 5 of the 54 patients were catheterized, although *only once*. The only excuse we have to offer for this is that in a hospital where there are 32 internes and where changes of interne staff occur several times a year, an accident of this sort cannot be entirely avoided. One of the 5 patients died. This was one of the early cases in the series, and careful study of the case record as well as an autopsy report fails to reveal an infection of the urinary tract as the cause of death. The other 4 patients did not develop any infection.

The management of a case may then be summarized as follows: When a case of injury to the spine is admitted to the ward, careful note is made of the neurologic complications including the condition of the anal and rectal sphincters. Occasionally even severe injuries in the cervical spine will escape injury to the reflex vesical path, although the patient suffers paralysis of his extremities. We have 2 such cases in our series, and Gurdjian[7] reports similar observations.

*Voluntary Emptying of the Bladder.* If the patient voids voluntarily and the urine is normal, the orthopedic surgeons continue in the care of the case. This is probably the type of case in which a concussion of the cord or other minor injury took place.

*Distention and Credeing.* If the patient fails to void within a reasonable time, more serious injury to the cord has very likely taken place, and either destruction of the vesical center or some other serious damage has occurred. When the bladder becomes distended, a urologic consultation is requested; the urologist tries to continue the policy of "watchful waiting." If the distention of the bladder is moderate and not painful, the patient is left alone. Stimulation by means of the mass reflex referred to previously is resorted to. In our hands this method has not been successful in emptying the bladder. If, on the other hand, the patient complains of some pain, Credeing of the bladder is tried. By this method one almost always succeeds in expressing some urine from the bladder. We have trained our orderlies and nurses in the wards to do this, and manual expression is carried out every four to six hours. It must be pointed out that although this is a satisfactory procedure, it must be resorted to *before* the distention becomes too marked. When the latter condition arises, manipulation of the bladder is very painful and not permitted by the patient.

*Distention and Overflow.* If there is no discomfort, and this is the most usual finding, the bladder is allowed to go on to distention until overflow occurs. We have waited twenty-four to thirty-six hours and more, and finally the bladder did empty itself.

Physicians who are unfamiliar with this "let alone" policy usually object with the statement "But you can't allow the bladder to become distended indefinitely." Invariably the answer is that "indefinitely"

means about twenty-four to thirty-six hours, and that in any event in our large experience we have never seen a bladder rupture from over-distention. The literature fails to reveal such an accident.

Once the bladder has succeeded in voiding some urine by overflow, it will continue to do so for several weeks to several months until automatic control is established. That this occurs in every case is conceded by all observers. Sir Robert Jones writes:

> The recovery of reflex automatic function in that part of the cord which lies below the level of the lesion may be expected whether or not the latter amounts to a complete anatomical division. The speed with which the reflex activity returns . . . depends upon the general condition of the patient. . . . In favorable cases in which the lesion is above the lumbosacral enlargement, the knee and ankle jerks may reappear two or three weeks after the date of injury. What has been said in regard to the reflex actions of the lower limbs applies equally to those of the bladder and rectum. These organs may develop the power of automatic evacuation at periodic intervals even when the lesion of the spinal cord precluded any possibility of recovery in regard to voluntary action.

In the case of patients whose bladder is allowed to empty by overflowing, special care must be given to prevent the formation of bed sores. Only one patient in our series developed bed sores, although our nursing facilities are very limited.

*Distention and Suprapubic Cystotomy.* There finally arises the type of case where either because Credeing was not instituted early enough, or because Credeing is very painful, or because of the pressure which the distended bladder produces upon the abdominal viscera there is marked abdominal pain and distress together with tympanites and vomiting; in other words the type of case in which the surgeon feels that "something must be done to empty the bladder." This is the type of case in which we do a suprapubic cystotomy. In the series here reported, two such operations were carried out without any succeeding complications, and the patients went home cured.

Before concluding the chapter on the management of these cases, the writers wish to mention only the management of the chronic type of case. These are patients who have survived more or less serious infections of the urinary tract, but who nevertheless have a residue of cystitis, pyelonephritis, and in some cases calculous pyonephrosis. They present special problems of interest only to the urologist. It is the authors' belief that many, if not all, of these cases will disappear when catheterization will be discarded as a method of treatment.

## ANALYSIS OF 54 CASES OBSERVED IN HARLEM HOSPITAL

Let us now study the 54 cases that were treated in Harlem Hospital in the past three years.

*Location of Lesion.* The cervical region was involved 17 times; the dorsal region, 11 times; the lumbar region, 27; and the coccyx, once. (The total according to these figures is 56, whereas 54 cases were really treated, but two of the cases had multiple lesions and are counted twice.)

*Frequency of Urologic Complications.* Of the 54 patients, 21 had definite urologic complications, that is retention of urine or incontinence.

*Sex.* Thirty-nine of the patients were males; 15, females.

*Mortality.* There were 20 deaths in this series, that is, a mortality of 37 per cent.

Of the 20 deaths, 13 of the patients had injuries in the cervical region. The other 7 deaths were due to internal injuries, fractured skull, etc. A more accurate cause for death cannot be given in all cases because they died within twenty-four or forty-eight hours, and autopsies were not always obtainable. Undoubtedly the fracture of the spine was a small or an unimportant factor in the causation of these 7 deaths, but inasmuch as either *x*-ray, or clinical examination, or examination by autopsy had proved injury to the spine,

it was deemed advisable to include all these deaths in the series. The striking finding thus far is that of the 17 patients who were admitted with injuries to the cervical spine, only 4 lived, an indicated mortality of 76½ per cent. Only one of these patients was catheterized. This case will be discussed again later when the deaths are analyzed in detail.¶

### THE DEATHS

We feel that the purpose of this article will have been lost unless a critical study is made of the deaths; if any of the deaths are due to causes which are remediable we should be aware of these facts and make every effort to obviate them.

*Group* 1. Seven of the patients died within twenty-four hours after admission. Their histories will now be summarized very briefly.

Case 1. Male, aged twenty-eight. History of having jumped from third story window. Examination revealed fracture of first and second lumbar vertebrae, also multiple fractures of the long bones of the lower extremities, probably also internal abdominal injuries. This patient died fourteen hours after admission.

Case 11. Male, age unknown. Found in an unconscious state. Examination revealed fracture of sixth cervical vertebra with complete paralysis of all the extremities. This patient died eight hours after admission.

Case 111. Male, aged twenty-four. Unreliable history states that patient became unconscious and fell. Examination revealed fracture of fifth and sixth cervical vertebrae and fracture of skull. This patient lived one day.

Case IV. Male, age unknown. Said to have jumped from third story window. Patient admitted to hospital in extremis. Examination revealed fracture and dislocation of the 1st lumbar vertebra, possibility of other injuries not excluded. Patient lived one one-half hours.

Case V. Male, age forty-four. Patient said to have fallen one flight of stairs while intoxicated. Examination revealed fracture of the skull and cervical spine and complete paralysis of all extremities. Slight distention of lower abdomen noted towards end (probably bladder). Patient lived one day.

Case VI. Male, aged twenty-nine. Found unconscious in the street; probably struck by automobile. Lived one day. Autopsy revealed a "crushing injury of the fourth cervical vertebra. The cord shows a pulpified area of hemorrhage in that region."

Case VII. Female, aged forty. Patient jumped from the fourth story to the street below. Examination revealed fractures of the skull and fifth lumbar vertebra, also multiple fractures of the bones of the lower extremities and the pelvis. The patient lived one day.

*Group* 2. Six of the patients lived for two days.

Case VIII. Male, age unknown. History of having fallen from the roof of a five story building to the roof of a two story building adjoining. Examination revealed fracture of the cervical spine with complete loss of sensation and motion from the level of the umbilicus down. Twenty-four hours after admission it was noted that the bladder was markedly distended. Thirty-six hours after admission the patient began to show incontinence of overflow. Patient died in forty-eight hours.

Case IX. Male, aged thirty-three. The history which was obtained from friends states that the patient fell while intoxicated. Examination revealed the presence of a compression fracture of the cervical cord and a complete paralysis of the lower abdomen and both lower extremities. The day after admission the patient had incontinence of overflow. He died in forty-eight hours.

Case X. Male, aged forty-six. Found unconscious. The first twenty-four hours the patient had retention of urine, then he voided voluntarily. Patient died in forty-eight hours; autopsy revealed crushing of fifth, sixth, and seventh cervical vertebrae with hemorrhage into the spinal cord substance.

Case XI. Male, aged twenty-three. Found unconscious. Said to have fallen from a height of 10 feet. Patient was incontinent throughout his stay in the hospital. He died in forty-eight hours. Autopsy revealed multiple fractures of the skull with hemorrhage into the epidural and subarachnoid spaces; fracture of the third cervical vertebra.

Case XII. Male, aged eighteen. Patient fell between a subway train and platform. Examination revealed multiple lacerations of the scalp and also multiple fractures of the

ribs and fracture of the dorsal spine; considerable abdominal tenderness probably due to internal injuries. Twenty-four hours after admission the bladder began to be distended. This distension increased until the patient died about forty-eight hours after admission.

Case xiii. Female, aged seventy-three. Injured in automobile accident. Although the diagnosis of fractured seventh cervical vertebra was made, the patient had no neurologic symptoms. She voided spontaneously. The patient lived for forty-eight hours. Autopsy revealed a complete fracture of seventh cervical vertebra.

*Group* 3. Patients living more than two days:

Case xiv. Male, aged sixty-three. Patient found in a hallway; states he was struck on the back of the neck. Examination revealed a complete paralysis below the level of the fourth rib. Moderate distention was present on third day. The patient voided voluntarily. Patient died three days after admission. Autopsy revealed a crushing fracture of the third cervical vertebra. The urinary tract was negative.

Case xv. Male, aged fifty. Found unconscious in an alleyway. Patient was bleeding from the ears and nose, and was found to have complete anesthesia from the level of the umbilicus down. After twenty-four hours the bladder was distended to the level of the umbilicus, then there was incontinence of overflow. Patient lived for four days. Autopsy revealed the presence of a hemorrhage into the brain and fracture of the dorsal spine.

Case xvi. Male, aged fifty-four. Admitted in shock with history of a fall from height of two stories to pavement below. Examination showed complete paralysis of both lower extremities, abdominal tenderness probably due to internal injuries, fracture of lower cervical spine. Patient voided on day of admission. On third day bladder became distended and remained so until death which occurred on the fourth day.

Case xvii. Male, aged forty-two. Admitted to hospital with history of having fallen from a fence, height unknown. Examination showed absence of abdominal reflexes, sensory and motor paralysis of both lower extremities. Bladder distended the first thirty-six hours, then incontinence of overflow. Patient died after five days; autopsy revealed fracture dislocation of sixth and seventh cervical vertebra with compression of the cord; fracture of the skull and laceration of the brain; no urologic findings.

Case xviii. Male, aged forty-two. Admitted with history of drinking for several days. Unable to say how he sustained injury. Examination disclosed anesthesia from the level of the nipple line with complete paralysis of the lower extremities. X-ray showed fracture of the sixth and seventh cervical vertebra and anterior dislocation of the third and fourth dorsal. Bladder distended; Credeing resorted to; started to void second day after admission and continued to do so. Patient died after six days. Autopsy was negative; urinary tract included. Permission not obtained for examination of skull and spine.

Case xix. Female, aged twenty-seven. Patient admitted with history of a fall from one story window while intoxicated. Examination disclosed multiple contusions and abrasions of the head and chest with signs of fracture of sixth, seventh, and eighth ribs. X-ray showed crushing fracture of the fourth and fifth lumbar vertebrae. Credeing of the bladder carried out throughout the hospital stay, in order to obtain evacuation of the bladder. Patient died after eight days from "internal injuries."

Case xx. Male, aged fifty-three. Patient was driving a truck in an intoxicated condition and collided with an automobile. On admission to the hospital there was a sensory paralysis from the level of the fourth cervical vertebra and complete paralysis of the lower extremities. On the day of admission he was catheterized because his bladder was distended. Then he developed incontinence and overflow. Ten days after admission he developed a decubitus ulcer on the sacrum. Repeated urine examinations at regular intervals, the last one three days before death, failed to show the presence of an infection in the urinary tract. The patient lived six weeks; the autopsy report was: "Narrowing vertebral canal at the sixth cervical vertebra due to crushing fracture of the body of the sixth cervical vertebra."

## SUMMARY

1. The literature on injuries to the spine has been reviewed with especial reference to morbidity and mortality statistics both in military and civil practice.

2. The pathologic physiology occurring in the spinal cord and in the urinary tract is described briefly.

3. Infection of the urinary tract is found to be the most important cause of death in these injuries. Catheterization of the bladder is accepted as the cause for infection in the opinion of almost all commentators.

4. The management of the urologic complications has been outlined, stress being laid upon the absolute necessity for the avoidance of catheterization. We are aware that some cases are not infected by catheterization, but we feel that this is a matter of luck rather than sound judgment. We feel that twenty years of clinical experience justifies our views on this subject.

5. An analysis is made of 54 cases treated in Harlem Hospital without a single infection in the urinary tract.

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# MEDICOLEGAL ASPECTS OF DEATHS ASSOCIATED WITH CHLOROFORM OR ETHER*

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KNOWLEDGE of the quantity of chloroform or of ether present in the organs of the deceased may often be of extreme importance from a medicolegal standpoint. This may be well illustrated by a case taken from the files of the Chief Medical Examiner's Office of New York City.

A woman, aged thirty-five years, was reported as having suddenly died in a physician's office. When the medical examiner arrived at the scene, he found the woman lying on a couch. No objective evidence could be found. The doctor was questioned about the manner of death. He claimed that this woman entered his office and told him that she had been aborted in another part of the city, some distance away. While walking along she began to feel ill, and entered the doctor's office for aid. While telling her story she suddenly collapsed and died before he could attend her. On further questioning the doctor maintained that he administered no anesthetic, and that he performed no operation of any kind.

The body was removed to the morgue where an autopsy was performed by Dr. Charles Norris, Chief Medical Examiner of New York City. The autopsy disclosed a perforation of the posterior vaginal vault, with the presence of about 300 c.c. of blood in the peritoneum, but there was no evidence of peritonitis. It was evident therefore that the cause of the death was due to hemorrhage brought on by the abortion. The all-important question, whether the abortion was performed in this doctor's office, or by someone else in another part of the city, remained to be answered. If large amounts of some anesthetic were present in her organs, it would indicate that she was in an anesthetized state at the time of death, and could not have walked the streets; on the contrary the abortion must have been done on the premises. In order to solve this question, parts of the brain, liver, lungs and stomach contents were submitted to my laboratory for toxicological investigation.

*Chloroform.* The results of the chemical analyses revealed fairly large amounts of chloroform in the brain, liver and lungs. No chloroform was found in the stomach contents, indicating that the chloroform did not enter the system by way of the stomach but by inhalation. In order to quantitatively determine the amount of chloroform present in the organs, the method devised by Gettler and Blume[1] was used. The quantitative analysis revealed 156 mg. of chloroform in 1000 gm. of brain, 110 mg. in 1000 gm. of liver, and 52 mg. in 1000 gm. of lung tissue.

Experience from scores of fatal chloroform cases has taught us that the chloroform content of the brain is more closely related to the physiological condition of the organism than the chloroform content of liver, lung or other tissues. It is for this reason that emphasis is placed on the chloroform content of the brain.

In order to decipher what physiological effect 156 mg. of chloroform in 1000 gm. of brain had on the individual in question, a comparison was made with our series of chloroform cases (Table 1) in the files of the Chief Medical Examiner's Office of New York City.

On looking over the figures of Table 1, we find that in the case of persons anesthetized for minor operations, the chloroform content is between 120 mg. and 182 mg. in 1000 gm. of brain. The case in question had 156 mg., indicating that the deceased person was under the influence of chloroform and therefore not able to walk; hence, the chloroform must have been administered on the premises.

It seemed important to investigate this problem further by means of experiments on animals, to determine how soon after chloroform anesthesia the individual is

* Contribution from the Chemistry Department of Washington Square College and Department of Forensic Medicine, Bellevue Medical College, New York University.

TABLE I

CHLOROFORM CONTENT OF THE BRAIN IN DEATHS ASSOCIATED WITH THE ADMINISTRATION OF CHLOROFORM

| *Case* | *History* | *Mg. Chloroform in 1000 Gm. Brain* |
|---|---|---|
| 1 | Suicide by inhalation | 432 |
| 2 | Suicide by inhalation | 480 |
| 3 | Suicide by inhalation | 410 |
| 4 | Suicide by inhalation | 390 |
| 5 | Homicide by inhalation | 372 |
| 6 | Homicide by inhalation | 384 |
| 7 | Homicide by inhalation | 374 |
| 8 | Anesthesia for abortion, shock | 162 |
| 9* | Anesthesia for abortion, hemorrhage | 60 |
| 10 | Anesthesia for operation on tonsils, shock | 136 |
| 11 | Anesthesia for minor operation, shock | 135 |
| 12 | Anesthesia for minor operation, shock | 145 |
| 13 | Anesthesia for minor operation, shock | 120 |
| 14 | Anesthesia for minor operation, shock | 122 |
| 15 | Anesthesia for abortion, shock | 182 |
| 16† | Anesthesia for minor operation, shock | 70 |

* Patient came out of anesthesia; died of hemorrhage.

† Patient died from shock during administration of chloroform; was not fully anesthetized.

again able to walk normally, and how much chloroform is present in the brain at varying intervals of time after the administration of chloroform. The present report is based on this work.

## TECHNIQUE OF EXPERIMENTS ON ANIMALS

Ten dogs were used in these experiments. After being tied to the animal board, they were anesthetized with chloroform, the open mask method being used. They were kept in a state of surgical anesthesia for definite periods. The mask was then removed, and the animals were allowed to recover spontaneously. Dog 6 was not allowed to recover, but was killed by excessive administration of chloroform. In dogs 5 and 7, the trachea was clamped while they were fully anesthetized. In order to investigate the chloroform content of the brain at various stages of recovery, the dogs were killed at stated intervals, as indicated in the chart. Painless death was obtained by injecting morphine and then exposing and clamping the trachea. Autopsy was then performed. The brain and lungs were removed, weighed and placed in the icebox in sealed containers until the analysis was started.

## RESULTS OF ANALYSES

The results, which are charted in Table II, indicate the following (all values calculated to 1000 gm. of brain tissue):

A dog killed by excessive administration of chloroform had 551 mg. of chloroform in 1000 gm. of brain tissue. Dogs killed while in chloroform anesthesia had 270 mg. and 284.6 mg. of chloroform in the brain. Dogs that were allowed to recover from the anesthesia showed a rapid decrease in the chloroform content of the brain, i.e.: after thirty-four minutes, 51.3 mg.; after fifty minutes, 33.3 mg.; after sixty-five minutes, 32.6 mg.; after ninety-five minutes, 23.6 mg.; after 102 minutes, 10.6 mg.; after 186 minutes, 5.6 mg., and after 190 minutes, 0.16 mg.

It was between thirty and forty-five minutes before the dogs looked normal and could walk normally. Dog 1 probably was killed just as he was beginning to return to normal. He had 51.3 mg. of chloroform in the brain. Fifteen minutes after dog 2 seemed normal, he had 33.3 mg. Thirty

five minutes after dog 3 seemed normal, he had only 32.6 mg. This is an indication that from fifteen to thirty minutes after the animal seems normal, the brain contains only from 30 to 35 mg. of chloroform in 1000 gm.

contain at the most from 30 to 35 mg. of chloroform.

The various points determined in our experimental work are vividly brought out by plotting milligrams of chloroform in 1000 gm. of brain against time in minutes

TABLE II
RATE OF DISAPPEARANCE OF CHLOROFORM FROM THE DOG'S BRAIN DURING RECOVERY FROM ANESTHESIA

| *Dog* | *Body Weight (kg.)* | *Weight of Brain (gm.)* | *Time in Minutes* | | | | | | | *Mg. of Chloroform in Brain at Death* | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | To Anesthetize | Until First Signs of Recovery | Until Placed on Floor | At Which Hind Legs were Still Ataxic | Until Dog Began to Look Normal | Until Death | Between Normal Appearance and Death | In Entire Brain | Calculated to 1000 Gm. of Brain |
| 1 | 8.5 | 80 | 15 | 6 | 8 | 19 | * | 34 | * | 4.10 | 51.3 |
| 2 | 5.7 | 75 | 15 | 5 | 17 | 30 | 35 | 50 | 15 | 2.50 | 33.3 |
| 3 | 6.3 | 69 | 15 | 4 | 6 | 13 | 30 | 65 | 35 | 2.25 | 32.6 |
| 4 | 6.9 | 70 | 15 | 6 | 10 | 75 | * | 95 | * | 1.65 | 23.6 |
| 5 | 9.4 | 82 | 12 | 5 | 10 | 35 | 45 | 102 | 57 | 0.87 | 10.6 |
| 6 | 5.2 | 52 | 15 | 5 | 6 | 30 | 42 | 186 | 144 | 0.27 | 5.2 |
| 7 | 11.3 | 60 | 15 | 4 | 8 | 25 | 30 | 190 | 160 | 0.01 | 0.16 |
| 8 | 7.6 | 60 | 15 | Trachea clamped while completely anesthetized | | | | | | 16.20 | 270.0 |
| 9 | 6.3 | 65 | 15 | Trachea clamped while completely anesthetized | | | | | | 18.50 | 284.6 |
| 10 | 7.6 | 68 | 7 | Killed by excessive administration of chloroform | | | | | | 37.50 | 561.5 |

* Morphine injected while animal was still ataxic.

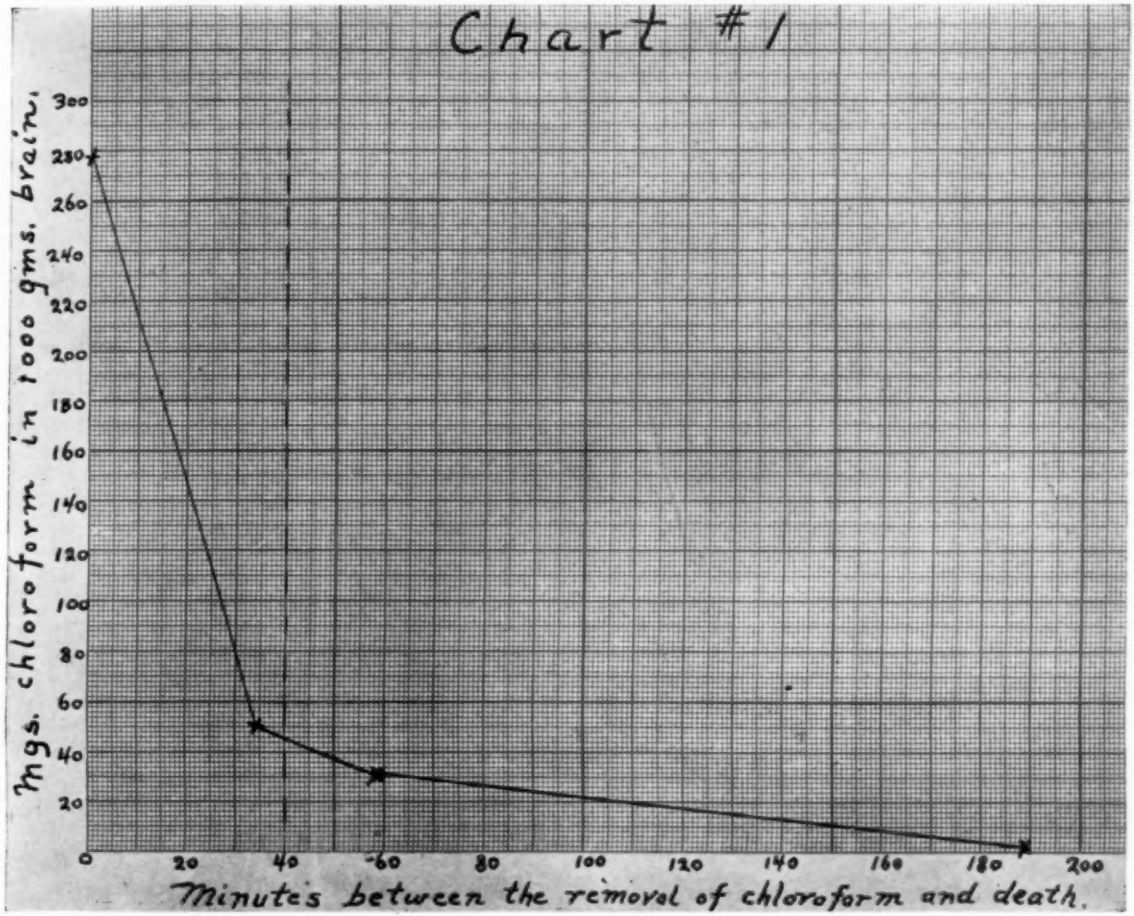
Chloroform content of human brain is best reported on 1000 gm. basis; in order to facilitate comparison, the chloroform in the dogs' brains was also calculated to 1000 gm. portions.

The lungs in some of the dogs were also analyzed. The values are not charted. We found that in the recovery stage the lungs contained only from 33 to 50 per cent of the amount of chloroform found in the brain.

The chloroform content of the brain of the deceased person in question was 156 mg. in 1000 gm. of tissue. On the basis of the determined figures, we hold that: The woman at the time of her death was still under chloroform anesthesia and not in a condition to walk. With 50 mg. of chloroform or thereabouts per 1000 gm. of brain tissue, she would have shown the first indication of normal coordination. If it took her only from fifteen to thirty minutes to get to the doctor's office, her brain should

(Chart 1). Attention might be called to the following points brought out by the plotted curve: When animals which have been in full chloroform anesthesia are allowed to recover, the chloroform content of the brain drops rapidly. In our experiments in thirty-four minutes it dropped from 278 mg. to 51.3 mg.; in fifty-eight minutes it was down to 33 mg. From this point on, the chloroform kept decreasing, but more slowly. After 188 minutes, only 2.7 mg. of chloroform in 1000 gm. were left (average values for dogs 6 and 7). In the chart, the vertical dash line at the forty-minute interval indicates the point at which the animals first began to appear normal again. Eighteen minutes after the

animals seemed normal, the brain contained only 33 mg. of chloroform in 1000 gm.




## ETHER

The problem of proving definitely the presence of ethyl ether, $(C_2H_5)_2O$, in human tissues has remained unsolved to the present time. There are no specific color or precipitation reactions for ether. Methods used by Nicloux,[2] Shaffer and Ronzoni,[3] Haggard,[4] and Spenzer[5] for the quantitative determination of ether are based on oxidation-reduction reactions. These are not specific qualitative tests for ether. Many organic substances, such as hydrocarbons, alcohols, aldehydes, ketones and organic acids, react as ether does toward these oxidizing agents. In medicolegal cases the pathologist or toxicologist has to rely on his sense of smell. When the body cavities are opened, he seeks the odor of ether. Proof of this kind is unsatisfactory and often misleading. For example, odors of various kinds may be encountered in body cavities and in organs which at times may simulate that of ether and at other times may conceal the odor of ether when it is present. The problem we set out to solve was the actual isolation of ether in pure form from the organs and its identification by determination of the boiling point.

Gettler, Niederl and Benedetti-Pichler[6] in their contribution on the isolation of ethyl alcohol from human tissues devised a specially constructed rectification flask. This apparatus was used at first in the attempt to isolate ether from tissue distillates. It served the purpose quite well. We succeeded in isolating droplets of

ether, proving the same by determinations of the boiling point.

It was found that certain structural changes in the rectification flask not only would facilitate the isolation of the ether, but would also allow the collection and measurement of practically all of the ether present in the tissues. The changes in the micro-rectification flask, and the method used for isolating the ether in pure form from the tissues is that of Gettler and Siegel.[7]

Table III
STUDY OF RECOVERY OF ETHER

| *Nmuber* | *Tissue Used 500 Gm.* | *Ether Added (c.c.)* | *Ether Recovered (c.c.)* | *Ether Lost (c.c.)* | *Percent Recovery* |
|---|---|---|---|---|---|
| 1 | Brain | 0.06 | 0.03 | 0.03 | 50 |
| 2 | Brain | 0.06 | 0.03 | 0.03 | 50 |
| 3 | Brain | 0.12 | 0.05 | 0.07 | 42 |
| 4 | Brain | 0.12 | 0.07 | 0.05 | 60 |
| 5 | Brain | 0.15 | 0.10 | 0.05 | 67 |
| 6 | Brain | 0.15 | 0.09 | 0.06 | 60 |
| 7 | Liver | 0.30 | 0.24 | 0.06 | 80 |
| 8 | Brain | 0.30 | 0.25 | 0.05 | 83 |
| 9 | Brain | 0.45 | 0.37 | 0.08 | 82 |
| 10 | Brain | 0.45 | 0.38 | 0.07 | 84 |
| 11 | Liver | 0.60 | 0.47 | 0.13 | 78 |
| 12 | Brain | 0.72 | 0.63 | 0.09 | 88 |
| 13 | Brain | 0.75 | 0.67 | 0.08 | 89 |
| 14 | Brain | 0.90 | 0.78 | 0.12 | 87 |
| 15 | Brain | 0.90 | 0.74 | 0.16 | 82 |

### RECOVERY OF ETHER FROM TISSUES

Five hundred gram portions of brain tissue and liver tissue were used. Varying amounts of accurately known quantities of ether were added to the tissues. All possible precautions were taken to avoid loss of ether due to vaporization. The method of Gettler and Siegel[7] was used to recover the ether which had been added to the tissues. The results are given in Table III.

Brain, liver and lungs of patients who died during or after ether anesthesia were analyzed for their ether content. The results are charted in Table IV.

Table III indicates that by our method as little as 0.06 c.c. of ether in 500 gm. of tissue can be isolated. It shows further that if tissues contain about 0.3 c.c. or more of ether in 500 gm., the yield of isolated ether is on the average 83.6 per cent (±5 per cent). If the ether present in the tissues is very small (cases 1 to 6), the percentage yield is much smaller (from 42 to 60 per cent). It will be noticed, however, that the ether lost in the procedure is extremely small (from 0.03 to 0.07 c.c.). This loss is relatively large on the basis of percentage. In cases in which the patient dies under full ether anesthesia or as a result of an accidental overdose of ether and in homicides by the administration of ether, the ether in the tissues is always higher than 0.3 c.c. in 500 gm. Therefore, in such cases, the yield is good and fairly constant. The direct measurement of the isolated ether can be used for the estimation of the ether actually present in the tissues.

The data in Table IV prove that the ether (in excess of 0.03 c.c.) present in tissues as a result of anesthetization can be isolated and measured. Recovered ether in amounts as small as 0.03 c.c. can be used for the determination of the microboiling point. The boiling points obtained on the specimens of isolated ether ranged from 34.5 to 35.1 c. These values compare favorably with the theoretical boiling

point of ether, from 34.7 to 35 c. The boiling point, therefore, can be used for identifying the ether. Other properties of identification of ether are: It is a thin mobile liquid; it is lighter than water, does not freeze in a solid carbon dioxide-acetone bath, has an ethereal odor, takes fire rapidly and burns brilliantly.

tissue. Animals while fully anesthetized with chloroform contained in the brain 270 mg. and 284.6 mg. in 1000 gm. of tissue. When an animal was in the stage of recovery, the chloroform content of the brain dropped very rapidly during the first thirty-four minutes, so that only 51.3 mg. in 1000 gm. of brain tissue was

TABLE IV
ANALYSIS OF CASES IN WHICH ETHER WAS ADMINISTERED

| *Patient* | *Comment* | *Tissue Used 500 Gm.* | *Ether Recovered* (c.c.)* | *Boiling Point of Isolated Ether* |
|---|---|---|---|---|
| M. R. | Died before fully anesthetized | Brain | 0.03 | |
| | | Liver | 0.01 | |
| | | Lung | None | |
| J. P. | Died after a few inhalations of ether | Brain | 0.09 | 34.8 |
| | | Liver | 0.08 | 34.9 |
| | | Lung | 0.02 | |
| E. W. | Died on table; no operation | Brain | 0.19 | 28.5† |
| T. J. | Died on table; no operation | Brain | 0.50 | 34.9 |
| | | Lung | 0.30 | 34.9 |
| | | Liver | 0.34 | 34.7 |
| W. O. | Died on table just after operation | Brain | 0.11 | 35.0 |
| | | Lung | 0.10 | 35.0 |
| | | Liver | 0.14 | 34.6 |
| M. J. | Anesthesia stopped 15 minutes | Brain | 0.37 | |
| G. W. | Anesthesia stopped 30 minutes | Brain | 0.21 | 35.1 |
| | | Liver | 0.22 | 34.9 |
| | | Lung | 0.14 | 35.0 |
| T. M. | Anesthesia stopped 70 minutes | Liver | 0.11 | 35.0 |
| | | Lung | 0.09 | 34.5 |
| W. W. | Anesthesia stopped 75 minutes | Brain | 0.00 | |
| J. L. | Anesthesia stopped 85 minutes | Brain | None‡ | |
| R. F. | Anesthesia stopped 4½ hours | Brain | None | |
| L. A. | Anesthesia stopped 7½ hours | Brain | None | |
| L. J. | Anesthesia stopped 16 hours | Brain | None | |
| R. R. | Anesthesia stopped 20 hours | Brain | None | |

* This is approximately 84 per cent of the amount of ether actually present in 500 gm. of tissue.

† This ether contained ethyl chloride, hence the low boiling point. In cases of this kind the ether is separated from the ethyl chloride by fractional distillation.

‡ This does not mean that no ether was present. It simply means that probably less than 0.03 c.c. of ether was present.

We are drawing no conclusions as to the amount of ether in the brain when a patient is fully anesthetized, because the data are still too meagre. Further research is in progress on this phase.

## SUMMARY

A dog killed by the excessive administration of chloroform contained in the brain 551.5 mg. of chloroform in 1000 gm. of left. From then on, the chloroform content decreased more slowly, and after 190 minutes there was only 0.16 mg. of chloroform present in 1000 gm. of brain. Animals appeared normal again after forty minutes. At this time they probably had about 45 mg. of chloroform in 1000 gm. of brain. Fifteen to thirty minutes after the animals seemed normal, the brain contained 35 to 30 mg. of chloroform in 1000 gm. In the stage of recovery, the lungs contained much

less chloroform than the brain. The chloroform content of the brain in humans during anesthesia was found to be between 120 and 182 mg. of chloroform in 1000 gm. of brain. In cases of suicide and homicide by chloroform, the brain was found to contain 372 to 480 mg. of chloroform in 1000 gm. of brain tissue.

A micromethod for isolating ethyl ether from human tissues is described. Amounts of ether as small as 0.06 c.c. in 500 gm. of tissue were isolated and measured. In tissues containing from 0.3 to 0.9 c.c. of ether in 500 gm., the recovery is 83.6 per cent (average). The recovery of ether is fairly constant and can be used for estimating the amount originally present in the tissues. Determinations of the microboiling point served to identify the isolated liquid as ethyl ether.

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# METALLIC POISONING SIMULATING THE ACUTE SURGICAL ABDOMEN

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DURING the past seven years I have seen 18 cases of heavy metal poisoning on the tour work of the Medical Examiner's office in New York City. No cases of salvarsan or neosalvarsan poisoning are included in this group. Twelve of these cases were suicidal, 2 were accidental, and in 4 it was never found out how the patient got the poison. Other Medical Examiners in the city had a similar series of cases during the same period, but I shall describe here only those cases autopsied personally. Mercury was the heavy metal causing death in 13 of the 18 cases, lead in 2, arsenic in 2, and "Paris green," a compound of copper and arsenic, in one. Sixteen of the patients were under observation in hospitals twelve or more hours up to several weeks before they succumbed. A definite clinical diagnosis was made in most cases (12 of 18). Of the remaining 6 cases no clear-cut diagnosis could be made until the autopsy was done. Before death 3 of the patients showed symptoms which led the medical attendants to suspect an acute surgical condition in the abdomen, but it is worthy of note that the patient was treated expectantly in each case and no operation was done, apparently because the surgeon felt that there was something unusual about each clinical picture. For this reason each case was referred to the Medical Examiner's office for investigation. Autopsy on these cases revealed gross pathological changes in the organs which indicated that the cause of death was poisoning by a heavy metal, the particular metal in question being determined by chemical analysis.

CASE 1. A Puerto Rican female, fifty-eight years of age, was admitted to the New York Hospital on June 2, 1931, by ambulance from her home. The history from her daughter, aged twenty-seven, was that the patient had had a sudden onset three days before of severe knife-like pain in the epigastrium which doubled her up and caused her to perspire. This was accompanied by retching and vomiting. Since onset the pain has been periodic every half hour or so lasting a few minutes. She has vomited everything she ate or drank and several times vomited blood. The bowels have been regular, the stools formed and tarry black in color.

On examination the House Surgeon described an obese white female in mild shock. The sclerae were possibly slightly icteric. The abdomen was obese and tender in the epigastrium. There was no rigidity or distention and the liver and spleen were not felt. No masses could be palpated. Her temperature was 100°F., pulse 100, respirations 22, and blood pressure 158/82. A blood count showed 5,000,000 R.B.C. and 75 per cent hemoglobin with 33,000 W.B.C., 94 per cent polymorphonuclear. The catheterized urine on admission was muddy and contained many W.B.C. and a few R.B.C. A high hot colon irrigation returned dark brown fluid with a few small fecal particles and some mucus. A guaiac test on this was 4 plus. The House Physician diagnosed peptic ulcer, with pyelitis or kidney stones, cholecystitis and appendicitis to be ruled out.

The same night an attending surgeon made the following note: "Do not believe patient has perforated ulcer. Other possibilities are (1) a vascular accident, (2) an intestinal loop such as twist or thrombosis, (3) acute pancreatitis or liver degeneration. At present she is three days along in her disease and with so few signs of peritoneal irritation I would be inclined to await developments." A portable *x*-ray of the chest was negative. Another blood count showed 31,000 W.B.C., 93 per cent polymorphonuclear. At this time the blood urea nitrogen was up to 87 mg. per 100 c.c. and a third count showed 26,000 with 88 per cent polymorphonuclear. The next day another

attending surgeon was of the opinion that the patient had cholecystitis or acute pancreatitis and advised observation. A specimen of vomitus was watery, greenish yellow and showed a 2 plus guaiac, and the fourth blood count showed 4,300,000 R.B.C., 65 per cent hemoglobin, 23,000 W.B.C., 86 per cent polymorphonuclear.

The patient was now exquisitely tender in the mid epigastrium, practically afebrile, and the abdomen continued soft and was not distended. One observer thought she might have a large gastric ulcer that had become adherent to the anterior parietal peritoneum after perforation. Consequently a fluoroscopic examination of the stomach was made with barium. No signs of ulcer or perforation were seen. On this day, the third in the hospital, the icteric index was 3.1 and the blood sugar 211 mg. On June 7, two days later, vomitus showed a 4 plus guaiac test and 5 to 6 R.B.C. per low power field. A guaiac test on the stool was faintly positive and microscopic examination showed a few R.B.C. A catheterized urine specimen contained a trace of albumin, a few granular casts, a few pus cells and R.B.C. On June 8, the seventh day in the hospital, the House Surgeon made the following note: "This A.M. vomited brownish coffee ground material showing 4 plus guaiac. This afternoon complains of severe epigastric pain. Patient seemed to be improving up to now, but at present is in shock. Her color is ashen gray and pale. She is in a cold sweat. Blood pressure 90/60. Her abdomen is soft, tender in the epigastrium, and not distended." The shock grew worse and the temperature, never above 100°F., gradually fell to 97°F. and she died on the eighth day in the hospital. An immediate postmortem chemical examination showed a urea nitrogen of 278, sugar of 441, and a $CO_2$ of 44 per cent.

Because the case was a puzzling one it was reported to the Medical Examiner's office for investigation. No one who had observed this patient had suspected poisoning by a heavy metal. We found the following gross lesions at autopsy: (1) Erosions and underlying necrotic hemorrhagic areas in the mucosa and submucosa of the dependent portion of the greater curvature of the stomach over an area 5 inches in diameter; (2) a hemorrhagic colitis; (3) an acute degenerative nephrosis fairly typical of a bichloride kidney. Chemical examination of the kidney showed mercury present in large amounts. These findings were reported to the District Attorney and police for investigation.

It is comparatively easy in looking back on a case after the patient has been observed and thoroughly worked up, and finally autopsied, to say this symptom and that finding ought to have led to the correct diagnosis during life. Certainly the patient just described did not present the usual picture of mercury poisoning. She at no time confided to her attendants that she had taken bichloride and they had no reason to be suspicious that she had been poisoned with homicidal intent. On admission to the hospital on the fourth day of illness she presented the picture of an acute surgical abdomen. No lesions of the mouth or gums were present such as we are apt to see developing in mercury poisoning. I believe the absence of gingivitis and ulcerating stomatitis in this case was due to the fact that the woman was fifty-eight years old and both upper and lower jaws showed marked senile atrophy of the gums and underlying bone. There were no teeth in her mouth. Further study in the hospital revealed evidence of severe renal damage with a diminished urinary output, although her appearance was not that of a chronic nephritic with uremia. These observations were not given sufficient weight in sizing up this case. That the medical attendants were aware of the kidney condition is shown by the fact that they made a postmortem blood chemical analysis.

Nor were the autopsy findings absolutely typical of bichloride of mercury poisoning. The gum and mouth lesions and a distinct ulcerating colitis were not seen. The hemorrhagic necrotic localized gastritis was very suggestive of heavy metal poisoning. The swollen, edematous kidney which on section presented to the naked eye a widened, pale, lilac colored cortex with indistinct markings, next to a very congested dark blue medulla, caused us to consider the case one of mercurial nephrosis. Marked fatty degeneration of

the liver was not found, although a yellow, greasy liver with blurred markings is mentioned in some books as a common finding in mercury poisoning.

Case II. E. E., an Italian vegetable dealer, fifty-three years old, walked into the clinic at St. Vincent's Hospital on October 9, 1931. He complained of generalized pain in the abdomen, inability to void, and nausea and vomiting. His illness began five days previously with sudden pain across the epigastrium and lower front chest on each side, which had come on shortly after the evening meal and was followed by vomiting. The pain had remained generalized throughout the abdomen and he had vomited off and on up to the previous day. He had had no bowel movement for the past two days but had always been constipated. Since onset his stools were formed, brown, not bloody or tarry. Before his present illness he had never had any gastro-intestinal symptoms.

On examination he appeared acutely ill and pale. His temperature was 96°F. and pulse 80. The skin was cold and clammy, pulse barely perceptible, and the patient showed evidence of "air hunger" with a fruity odor to his breath. The respiratory rate was 28. The sclerae were jaundiced slightly. His abdomen was not distended but scaphoid and there was rigidity in the right upper quadrant. Fine rales were heard at both lung bases posteriorly. The blood pressure was 72/50. A blood count showed 14,000 W.B.C., 55 per cent polymorphonuclear. The patient was given an enema which returned brown fluid with small particles of fecal matter. He was catheterized and only a few drops of thick cloudy yellow urine were obtained which microscopically showed a number of pus cells and occasional R.B.C. Blood chemistry soon after admission showed N.P.N. of 81, creatinine 2.3, sugar 300 mg., and $CO_2$ of 21 per cent. The icteric index was 43. The House Surgeon's provisional diagnoses were: "(1) shock, acidosis, (2) ruptured appendix or empyema of the gall bladder, (3) pancreatitis, (4) acute nephritis with early uremia secondary to a toxic process." The Attending Surgeon then saw the patient and noted: "The abdomen is scaphoid, local tenderness and rigidity are marked in the R.U.Q. There are no signs of acute appendicitis. The sclerae and skin are jaundiced. The blood count is not in conformity with an acute surgical abdomen. This patient should be treated expectantly until the anuria has been relieved, or until indication for operation is more clear." His provisional diagnosis was: (1) chronic cholecystitis with stones, and (2) anuria—cause not understood. An attending physician then examined the patient and thought he might have a ruptured adherent viscus with reflex anuria. A genitourinary surgeon next saw the man and noted: "A flat genitourinary *x*-ray plate is negative. There is nothing f. lt in the loins. Rigidity seems high and in the front of the abdomen."

The evening of the day of admission the patient several times passed small amounts of bright red blood by rectum. Later that night his abdominal pain disappeared, his blood pressure dropped to 56/40, and at 7 A.M. the day after admission he suddenly ceased to breathe, having been under observation in the hospital only twenty-two hours, the seventh day since the onset of his disease. The House Surgeon considered the cause of death to be shock, acute pancreatitis and acute nephritis, but several of the other men who had observed the patient were not satisfied with this diagnosis and were unwilling to make a definite one. None of them, however, suspected a heavy metal poisoning. Because of the uncertainty of the cause of death and since the patient had been in the hospital less than twenty-four hours the case was reported to the Medical Examiner.

At autopsy we found a grayish blue discoloration of the gums around the lower lateral incisors: a lead line. There was slight jaundice of the skin and sclerae, and mucous membranes of the conjunctivae, lips, tongue and mouth were very pale. The liver showed marked fatty degeneration. Petechial hemorrhages were found in the areolar tissue at the root of the lungs, on the heart, in the mesentery, under the pleura, and under the mucosae of the duodenum and rectum. There was a membranous colitis and proctitis. The contents of the small gut were tarry. A horse-shoe kidney was found which on section was pale, edematous, and showed indistinct markings with a swollen opaque cortex. All the organs appeared anemic. The cause of death was noted as: "Fatty degeneration of the liver. Acute membranous colitis, heavy metal poisoning." Bone, liver, and kidney were sent to the chemist, who reported arsenic and mercury absent in liver and bone, but lead present in bone—(12.8 mg. in 285 gm.) and not in the liver. The kidney was not tested

for lead since if no lead is found in the liver it is unusual to find it in the kidney.

Lead is probably the most important of the metallic poisons.[1] Acute lead poisoning is rare. The type we usually see is similar to the one just described, a typical case of chronic lead poisoning. This type of poisoning is important both in industry and outside of it. So called "epidemics" of lead poisoning have occurred from drinking water contaminated by the lead in the pipes through which the water flows. Other cases result from drinking beer brewed in lead lined vats or wine contaminated by lead. Lead may get into food when the food is cooked in lead vessels. Vegetables and fruit may absorb enough lead to cause harm when eaten if they are sprayed with an insecticide containing lead arsenate. On being absorbed lead is probably transported in the blood stream in the form of colloidal lead phosphate, and deposited in the tissues, especially the bones, as tertiary lead phosphate. As long as the lead remains stored in the bones no symptoms of poisoning develop. It has been shown that as long as there is a positive calcium balance and a normal pH of the blood, lead remains in the bones. However, when a condition of alkalosis or acidosis obtains, it is thought that lead is dissolved out of the bones and re-enters the circulation coming into direct relation with vital tissues such as liver, kidney, and bone marrow. This may explain why a sudden appearance of symptoms of lead poisoning may occur in an individual who has not been exposed to lead absorption for some time.

In the case just described the importance of the line in the gums about the lower lateral incisors was not realized. However, the kidney condition, as evidenced by the laboratory findings and anuria, was fully appreciated, but its cause not understood by the surgeon. The patient did not look like a chronic nephritic with uremia on physical examination. The presence of jaundice, acute recent onset of abdominal pain and vomiting, and the fact that the patient showed local tenderness and rigidity in the right upper quadrant confused the clinical picture still more. Had the patient lived longer, further examinations of blood smears most probably would have shown marked basophilic stippling of the red cells, an important diagnostic sign of lead poisoning. After autopsy the best conclusion we could come to, was that the case was one of heavy metal poisoning. We did not know what the metal was until the chemical analysis had been made.

[1] Webster. Legal Medicine and Toxicology. 1930, p. 439, et seq.

Case III. J. F., a seventy-six year old white male, leather worker, was found on the floor of a public wash-room in an office building by some friends. About fifteen minutes before this he had had a quarrel with a business associate. An ambulance was called from St. Vincent's Hospital. The ambulance surgeon found him in severe pain but perfectly conscious. He had vomited and the interne noted a few small particles of wet, pale blue-greenish substance in his mouth. On admission to the ward on February 27, 1934, he complained of severe epigastric and precordial pain, vomiting two hours after his last meal (it was then 3:45 P.M.), chills and diarrhea. His statement of the present illness was that he had been seized with a sudden sharp pain in the xiphoid and precordium about noon. He vomited and he felt he was about to die. He vomited several times more and then had some watery bowel movements with no fecal masses. In 1925 he had had an operation for ulcer of the stomach but since that time there had been no symptoms referable to the gastrointestinal tract. During the past few months he had noticed some shortness of breath on exertion but no other cardiac symptoms.

On examination his temperature was 98°F., pulse 96 and regular, respirations 28, and blood pressure 110/70. He was in pain, orthopneic, forehead, face and neck were cold and clammy to touch, and there was slight cyanosis of his extremities. The abdomen was soft, not distended, and was tender to pressure in the epigastrium. A midline operation scar from xiphoid to navel was the site of a medium sized

ventral hernia. The heart sounds were distant, but no murmurs or friction sounds were heard. His chest was emphysematous which might explain the distant quality of the heart sounds. At this time it was thought the patient might have either an early intestinal obstruction, acute gastroenteritis or arteriosclerotic heart disease. A blood count showed 17,000 W.B.C., 71 per cent polymorphonuclear. The non-protein nitrogen of the blood was 42, the sugar 173, and the $CO_2$ 48 per cent. Vomiting of a watery fluid containing a few pale greenish particles continued, and he had frequent watery stools containing mucus shreds which on standing settled out into a pale green sediment. No blood was noted either in the vomitus or in the stool. An attending surgeon now made the following note: "Abdomen is soft throughout. There is well-marked and well-defined tenderness a little to the right and above the umbilicus. There is an old upper right rectus scar and hernia. Because of the character of the pain, site, tenderness, absence of rigidity, frequent vomiting and leucocytosis without fever my impression is acute pancreatitis." At 10 P.M. that night the patient had several shaking chills; his body was cold and moist. His temperature rose to 100.2°F., pulse dropped to 60, and the blood pressure fell to 40/0. His pain now was substernal. He became extremely anxious. No friction rub was heard over the heart and the heart sounds were noted as distant and regular. No murmurs were heard. Fine rales were present at the bases of both lungs posteriorly and in the axilla. The patient died the following morning having been in the hospital fourteen hours.

The House Surgeon, puzzled, was undecided whether to sign the case out as coronary thrombosis or report it to the Medical Examiner as one of acute gastroenteritis of unknown origin. The reason he decided to turn the case over to us for investigation was the fact that he had been impressed with the frequent watery stools with a pale green sediment, something he had never seen before.

Here is a summary of what we found at the autopsy. The body was that of a thin adult white male appearing younger than the stated age of seventy-six. The stomach showed a hemorrhagic membranous gastritis with abundant tenacious mucus mixed with small pieces of Paris green clinging to its mucosa. The mucosa, wherever it was in contact with the undissolved chemical, was necrotic. There were flame-shaped hemorrhages on the papillary muscles of the left ventricle and both sides of the interventricular septum beneath the endocardium, often seen in arsenic poisoning (Paris green is a compound of copper and arsenic). The small and large intestines likewise contained Paris green mixed with a watery fluid and a lot of mucus. Their mucous membranes were extremely swollen and edematous and showed a few small hemorrhages. An interesting finding was the presence of advanced atherosclerosis of the coronary arteries, heart valves and aorta. In the anterior descending branch of the left coronary artery there were calcification and atheromatous ulceration with an old thrombus which had become recanalized. There was a large fibrous scar in the myocardium of the left ventricle near the apex. The other viscera showed nothing especially characteristic of heavy metal poisoning. Chemical analysis of the stomach contents revealed arsenic and copper in large amounts.

Obviously, here we have a case of acute gastroenteritis caused by Paris green. On admission because of a history of abdominal operation for ulcer of the stomach, epigastric pain and persistent vomiting it was suspected that the man might have an acute intestinal obstruction. This diagnosis was soon discarded, however, in favor of the diagnosis of acute pancreatitis, because his abdomen was soft and not distended, and a watery persistent diarrhea developed, the stools somewhat resembling the nature of the vomited material. This fact is the important point in the case, i.e. watery stools and watery vomitus appearing grossly somewhat similar, and both containing pale green sediment. When enough arsenic had been absorbed from his stomach and intestines the patient went into collapse. Because he complained of substernal pain and appeared to be in severe shock, the interne treated him as a coronary occlusion for several hours until he died. We have seen many patients with coronary occulsion who have had an attack of vomiting soon after the onset of their pain but they vomit only once or twice, usually the

food eaten at the previous meal. They do not have a persistent watery diarrhea as seen in food, or other kinds of poisoning. In passing it is interesting to note that at autopsy this man had evidence in his heart of a former coronary occlusion.

Case IV. On admission to the hospital this case was not diagnosed as an acute surgical abdomen. However, because of its interest and the unusual clinical picture presented, I hope I will be pardoned for including it among our cases. Under different conditions this patient might very well have been thought to have had an acute surgical condition needing operation.

A white female housewife, thirty-two years of age, was admitted from her home to the New York Post Graduate Hospital December 17, 1930. The chief complaints were vomiting, headache, epigastric pain and diarrhea for five days. The patient was semi-comatose and irrational on admission. The husband stated that she was very religious and did not eat meat for supper the night of the onset of her present illness. She ate cheese instead, and was the only one of the family to do so. About two hours after the evening meal the patient became nauseated and vomited several times during the night. The family doctor was called in and treated the woman several days, when on the fourth day of the illness, he found that a urine specimen contained 4 per cent sugar. Insulin was given and there was a temporary improvement in the patient's condition. The next day she had diarrhea and became semi-comatose and on the sixth day of her illness she was sent to the hospital by her family physician as a case of diabetic coma. Her past history was unimportant except that she had had a "nervous breakdown" two years ago but she was not "sent away."

A resume of the House Surgeon's examination on admission follows: "Temperature 98.8°F., pulse 110, respirations 20. Patient is semi-comatose, mildly irrational and dehydrated. Her pupils are small, her face flushed and her lungs and abdomen are negative. A catheterized specimen of urine showed 30 W.B.C. per high power field and many granular casts per low power field. Three plus albumin was found, but no sugar. Blood pressure was 50/?. A blood sugar was 87 mg. per 100, urea nitrogen 36, and a $CO_2$ 44 per cent. The House Physician considered the following diagnoses: (1) food poisoning, (2) diabetic coma, (3) hysteria, (4) brain tumor. A blood count showed 4,900,000 R.B.C. with 90 per cent hemoglobin. Soon after admission the patient passed several brown soft stools with mucus, and later she passed liquid stools with blood and mucus. Her condition became gradually worse and she died eleven hours after entering the hospital.

The attending physicians were unable to make a definite diagnosis so the cause of death was reported to the office of the Medical Examiner as "unknown." At a partial autopsy we found a slightly enlarged liver. The surface was yellowish, mottled with small indistinct yellowish-pink areas. The organ cut easily and the cut surface was greasy, flabby and yellow-pink in color. The usual liver markings (the lobules) could not be distinguished. The body of this patient was not jaundiced. The gall bladder and bile ducts were natural. The stomach and intestines contained abundant mucus and small amounts of semi-fluid material resembling spinach. The mucosa of the stomach and jejunum was edematous and wet. The mucosa of the ileum was somewhat drier and appeared more normal. No hemorrhages, exudate, ulcers or signs of inflammation were found in the gastrointestinal tract. The large gut contained a very small amount of gray-brown soft fluid material. The kidneys showed acute fatty and parenchymatous degeneration of the cortex and were extremely flabby. Other abdominal organs were natural looking. We suspected at this time a poisoning of some kind, such as arsenic or phosphorus. This was confirmed by chemical examination of the liver and kidney. Both organs contained very large amounts of arsenic. Our findings were reported to the police and District Attorney for investigation as to how this woman got the arsenic. Incidentally this was never found out.

## SUMMARY AND DISCUSSION

We have reported in some detail 4 cases of heavy metal poisoning which clinically appeared at first to require immediate operation for the relief of an acute abdominal condition. On further study in the hospital they presented a puzzling picture. Fortunately each patient was therefore treated conservatively. The true

nature of these cases was not revealed until an autopsy had been done. The striking symptoms in each case on admission were severe abdominal pain and vomiting. For this reason the case was sent to the surgical service. Routine examination by the ward physician resulted in a number of "provisional diagnoses" including acute appendicitis, ruptured ulcer, acute pancreatitis, acute cholecystitis, kidney stones, intestinal obstruction, empyema of the gall bladder, coronary thrombosis, and acute gastroenteritis. On further study, all these cases showed a soft abdomen which was not distended and in which no mass could be felt. It is important to note that none of the patients had temperature. In one case the vomiting was persistent and frequent, and a watery diarrhea soon developed. The stools looked very much like the material vomited and were fluid, copious, and contained pale greenish-blue flakes. In the other cases the stools were abnormal in that they showed either fresh blood or a strongly positive guaiac (in the absence of hemorrhoids and other rectal lesions). The vomitus in 2 cases showed a positive guaiac reaction and microscopic R.B.C., and one patient vomited "coffee ground" material soon before she died. A questionable jaundice of skin and sclerae (in a Puerto Rican) was described in one case, but the icteric index was found to be within normal limits. Another case showed only a slight jaundice clinically, with an icteric index of 43. Severe kidney damage was evident from a study of the urine and blood chemistry in 3 cases. The other patient lived only fourteen hours after the onset and did not void during this time. Although none of these patients looked on inspection like a chronic nephritic with uremia, the urine showed W.B.C., R.B.C., casts, and albumin and was scant in amount. The N.P.N. and urea in the blood were elevated far beyond normal limits.

It is a dangerous thing to make generalizations. Nevertheless we think a study of these cases illustrates that when a patient presents the picture of an acute abdomen as evidenced by severe abdominal pain and frequent vomiting, but physical examination reveals no rigidity, distention or mass, and there is no fever, intensive study of the vomitus, stools, urine, and blood chemistry is indicated. It is worthy of note that all the patients who survived more than twenty-four hours after the onset of abdominal pain showed marked evidence of renal impairment. In each case it was possible to make the diagnosis of heavy metal poisoning at autopsy by the gross lesions found.

Note: Thanks are due to the New York Hospital, St. Vincent's Hospital, and the New York Post Graduate Hospital for permission to use their clinical records, and to Miss Story, of the Medical Examiner's office, who kindly collected these cases from our files.

# RELATION OF THE PHYSICIAN TO WORKMEN'S COMPENSATION INSURANCE

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THE Workmen's Compensation Act is usually enforced in the following way: A referee, who goes about over the state, holds a hearing at the most convenient place for the parties concerned. The proceedings are conducted with much less formality than in the regular courts, but a stenographer takes a record of all the testimony. If the plaintiff cannot afford the services of a private lawyer, an attorney in the service of the Industrial Commission represents the plaintiff without charge. The decision of the referee is final if both parties are satisfied, but either party may appeal to the Industrial Commission who may sustain or reverse the findings of the referee. The case may also be appealed from the Commission to the Supreme Court of the state. Testimony is taken only by the referee; the higher tribunals act only on the written record.

The most important evidence before the referee is usually furnished by physicians who give opinions as to the nature of the illness and its relation to trauma or other factors concerned in the occupation of the plaintiff. The clinicians who testify fall into three groups: Physicians consulted by the plaintiff who have taken care of him during his illness; physicians who have examined the plaintiff at the request of the defendant insurance company representing the employer; and physicians called by either party to testify as experts on the testimony presented. The latter group have usually had no direct contact with the plaintiff. In cases where a postmortem examination has been made the pathologist also testifies, and the pathologist may also be called upon for a report upon the diagnosis of a tumor or other lesion in which trauma has been alleged to play a role.

When workmen's compensation laws were first enacted, awards were made only for the known occupational diseases and for the direct effects of trauma or other injury; but there has been a widespread progressive tendency to include more cases in the compensative group. This broadened interpretation of compensative disease now includes all instances in which trauma or other injury is held to be a contributory or aggravating factor, and all cases where physical strain incident to the occupation is held to have had an unfavorable influence upon the course of any natural disease which the plaintiff had at the time. These new interpretations have had the effect of making the law indefinite, since the decision in such cases rests not upon facts but upon opinions. The degree to which a previous illness must be aggravated by injury or strain before the case becomes compensative is left to the judgment of the referee or the commissioners, and there is a great deal of inconsistency in the decisions that are being made at present. This is not surprising since the commissioners and referees have seldom had any medical training, and the contradictory medical opinions must be very bewildering to them.

Illustrations will next be given of the various types of cases that have come under my observation as a pathologist, with the decisions that have been rendered.

1. *Cases in which death was due entirely to trauma.*

Case I. A laborer fell under a truck and a rear wheel passed over his body, crushing the chest and causing rupture of the lungs and the heart. The postmortem examination revealed no disease aside from the traumatic lesions. The death claim was not contested.

There are a great many cases of this general type, and they are seldom contested when trauma is the obvious cause of death.

2. *Cases in which trauma is a contributory cause of death.*

Case II. A diabetic, fifty years old, sustained a severe injury to the left side of his body. The soft tissues were bruised and discolored, and there was a small piece of bone detached from the crest of the left ilium.

Diabetic coma developed and death occurred about one week after the injury. This case has not been decided, but trauma is obviously an important contributory factor.

Case III. An elderly diabetic developed gangrene of the great toe which extended up the leg and resulted in his death. It was alleged that the toe had been injured but from the evidence presented injury was not distinguished from primary gangrene. Compensation was awarded.

Case IV. A young man was struck on the chest in the region of the sternum by a large box which he was handling. A severe pain developed immediately and continued until his death. There was no physical evidence of bruising. At postmortem about one month after the injury, massive leucemic infiltrations were found around the bodies of the thoracic vertebrae and an area of the lower part of the sternum was found completely destroyed by leucemic infiltrations. Compensation was denied by the referee.

Case V. An elderly man fell on the sidewalk and sustained a fracture of the neck of the femur. It was claimed that he tripped over something on the sidewalk. A generalized adenopathy was present at the time of the injury and the subsequent course was that of Hodgkin's disease. At the postmortem several months later generalized Hodgkin's disease was found. The femur at the site of the fracture was extensively replaced by Hodgkin's tissue. Compensation was awarded.

Case VI. A man about fifty years of age had been complaining of cardiac pain throughout the day. In the late afternoon he was found dead at the foot of a stairway. The postmortem revealed coronary thrombosis and a fracture of the skull. Compensation was denied by the referee.

3. *Traumatic appendicitis.*

Case VII. A young man, while cranking an automobile, was struck by the crank in the lower right abdomen. Within a few days signs of appendicitis developed. Appendicitis was found at operation and the patient died of peritonitis. Compensation was awarded.

4. *Traumatic psychosis.*

Case VIII. A man known to have neurosyphilis was found unconscious near his overturned automobile. He had been reprimanded by a patrolman for erratic driving about three hours before the accident. A few superficial bruises were noted. He passed from coma into a severe psychosis which persisted until his death several months later. At postmortem extensive neurosyphilis was demonstrated. No traumatic lesions were found. Compensation was awarded.

5. *Trauma and neoplasms.*

Case IX. A laborer developed a basal cell carcinoma of the face. It was brought out in the evidence that he had been struck by a lump of coal at the site of the lesion a few months previously, but there was no medical observation of the lesion during this period. Compensation was awarded.

There are now a number of cases in which the courts have ruled that various neoplasms are of traumatic origin.

6. *Heat stroke.* When the clinical evidence is convincing these cases are not contested. Disputes arise in cases in which the patient has another disease and heat is a possible contributory factor.

Case X. A chronic alcoholic collapsed while at work on a hot day. His temperature was moderately elevated and he did not regain consciousness. The postmortem revealed a marked fatty metamorphosis of the liver. Compensation was awarded.

In a workman with any chronic disease there is lessened resistance to heat and it may be argued that his condition may be unfavorably influenced by temperatures that would not affect a normal man.

7. *Carbon monoxide poisoning.* It is easy to establish this diagnosis by chemical or microscopic examination of the blood from an unembalmed body. However, when the body has been embalmed a positive diagnosis cannot be made. The type of case contested is usually a man with a serious heart lesion found dead in a garage. In the embalmed body carbon monoxide cannot be excluded.

Certain forms of chronic encephalitis are now attributed by some physicians to the late effects of carbon monoxide poisoning; but the only well-established chronic lesion from carbon monoxide is lenticular degeneration.

8. *Cardiac disease aggravated by physical strain.* Recent decisions by Industrial

Commissions have made it possible to present a plausible claim in any case of chronic cardiac disease in which decompensation or a cardiac attack develops while the employee is engaged in heavy physical work.

Case xi. A milk-wagon driver, forty years of age, had worked regularly for several years. Each morning he would load his truck with full milk cans from a freight car. The loading required twenty to thirty minutes of fairly hard work. One morning he dropped dead just after the loading was completed. The postmortem revealed severe coronary sclerosis but no thrombosis. Compensation was awarded.

Compensation was also awarded in a case with a somewhat similar history in which coronary thrombosis was found at postmortem; and compensation has been denied in some cases not very different from those just cited. There have also been decisions favorable to the plaintiff in cases where cardiac decompensation developed after somewhat strenuous physical work, even though the patient was accustomed to do work of this kind.

There is obviously no way of determining to what extent physical exertion shortens the life of a man with chronic cardiac disease. Full compensation is often awarded in those cases where the cardiac attack develops while the man is on duty, but no award is made if the attack develops while he is not working.

9. *Cancer of the stomach caused by a burn.*

Case xii. A workman sustained a fairly severe burn of one hand and arm which, however, subsequently healed. Shortly after the accident symptoms of a gastric lesion developed and many months later the patient died of carcinoma of the stomach. It was maintained by physicians testifying for the plaintiff that the toxemia from the burn caused a gastric ulcer and that this subsequently became malignant. Compensation was denied by the referee but his decision was reversed by the Commission.

Wierd decisions of this kind are not infrequent; and they demonstrate clearly that chimerical speculation often prevails over sound medical thinking. The physicians themselves are, however, chiefly to blame for these decisions since the Commissioners always base their decisions on opinions furnished by physicians.

In the hearings before the referees the contradictory opinions expressed by physicians tend to weaken public confidence in the honesty of our profession. Too often it is apparent that the doctor's opinion is influenced by his interest in the outcome of the hearing; and we now have professional expert medical witnesses whose services may be secured by either party.

There are ways in which workmen's compensation laws might be improved so that they would operate more justly:

1. A well-trained honest physician should be appointed to the Industrial Commission in each state.

2. Full compensation should be given in all instances in which a normal person dies as a result of trauma or other injury as is done at present. Public opinion demands this compensation for the family of the deceased, and it is eminently just.

3. No special compensation should be allowed for cases in which trauma or physical strain is merely a contributory cause of death. At most this merely shortens the life of the individual to an undetermined extent. It is not comparable to the death of a healthy man from traumatism. The family of the deceased are just as much entitled to compensation when the worker with coronary disease develops the fatal attack at home as when it occurs during working hours; yet at the present time no compensation is allowed in the former and full compensation in the latter.

If these cases were excluded much expensive litigation would be avoided and funds would be available from what employers pay at present to give a definite sum to the family of every employee who dies, regardless of the cause of death.

The practice of allowing full compensation in cases of chronic heart disease, diabetes, hypertension, etc. is one important reason why older men find it difficult to secure employment. The employer knows there will be more death claims among the older group. A uniform compensation for all death cases would remove this difficulty.

4. Disabilities arising out of injury should be handled as they are at present.

# THE RELATIONSHIP OF ORGANIC DISEASE TO INJURY*

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IN medical literature until recently there has been but little systematic correlation of the relationship of latent organic disease to injury, although its existence is well known and any surgeon, especially one who deals with traumatic cases, has at some time or another encountered patients in whom healing of even trivial injury has been delayed, or caused severe complications (sepsis, etc.) ending in death. Subsequently a latent organic lesion or "extra-accidental" factors are found as the primary cause.

The possibility of pre-existing disease causing aggravation of an injury, or the possibility of claims made by an injured person as to an accident causing the organic lesion, is of tremendous importance to the industrial surgeon, and is a problem which he must constantly keep in mind.

In large industrial concerns this problem is attaining added significance, so that at the time of employment problems of possible industrial disease or accident in each individual are considered before the employee is given his job. Factors of race, age, sex, fatigue, previous health and, above all, individual idiosyncrasies or predisposition, play important roles in the causation of industrial accidents. It is for this reason that most of the large industries both here and abroad have well-organized medical staffs with *x*-ray and pathological laboratories and technical assistants, capable of performing a complete physical survey of a patient. Unfortunately, however, like so many well-meaning sociological endeavors, the industrial surgeon, who is on a salaried basis, tends to regress, a result of the destruction of individual incentive, and the type of medical service rendered is considerably deteriorated. To the credit of many of these men, however, it must be stated that they have rendered a high type of medical care to the industrial worker and, in some instances, have made noteworthy contributions to the industry with which they are connected as well as to the literature of industrial medicine. An excellent example is the research carried out by the medical department of the German dye industry in the causation of bladder tumors in certain workers, during the production of some of the coal-tar derivatives.

The industrial surgeon knows that it would be extremely rash to place an individual suffering from anemia or other blood dyscrasia at work in a plant where benzene is used, in any considerable amount, or where *x*-rays or radioactive substances enter into the manufacturing process. Similarly, individuals suffering from hypertension, nephritis, gastrointestinal lesions, as well as alcoholics, should not be employed where lead is worked.

Youth, as has been shown by Alice Hamilton, is much more susceptible to the so-called munition poisons (T.N.T., D.N.P., etc.) than are the mature. As an almost general rule females are more susceptible than males to all occupational diseases, with the exception of rheumatism and hernia. Susceptibility in healthy individuals varies greatly and constitutes one of the most troublesome factors in industrial medicine. The only safeguard which the plant physician may employ is to keep a careful watch on new employees, examining them every few days if that seems expedient. Thereafter periodic examination of every employee should be made, preferably every three to twelve

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months, depending on the nature of the industry and the individual idiosyncrasy of each employee.

Psychic factors in the injured worker must not be overlooked. It is true that many workers are entirely unaware of some organic disease within their body and are just as capable as their fellow workers of doing a day's work and earning full pay therefor. Only when a sudden injury occurs and he is no longer capable of performing his duties does the patient realize that something is wrong with him. It is, therefore, but natural that he should blame the accident for his resulting impairment. Such individuals frequently say that they would gladly give up their compensation moneys if their *status quo ante* were assured. On the other hand, there undoubtedly are a great number of workers who deliberately use all the resources at their command—often aided and abetted by dishonest lawyers and physicians—to make most fantastic claims for minor accidents. Professional "accident rings" are too well known to require further comment.

With all these possibilities in mind, the industrial surgeon should and must employ immediately the most complete technical aid in order to determine or exclude a pre-existing lesion or the source of a possible complication and to return the injured worker to his job, with a minimum of loss in time and cost.

Frequently claims are made by the relatives or the estate of an injured or killed worker that the injuries, often trivial in nature, are the cause of a severe disease, or even caused the death of the employee, when, in fact, such diseases have existed latently long prior to the alleged accident.

Recently a young steel worker developed hyperthyroidism for which thyroidectomy was advised. Following a partial thyroidectomy the patient died. His estate claimed death benefits from the employer, alleging cyanide poisoning from fumes in the steel plant as the cause of the hyperthyroidism.

In traumatic injuries the question of secondary infection by pus or gangrene-producing bacteria and of latent constitutional disease, particularly tuberculosis, syphilis and diabetes, must always be borne in mind. The former, for obvious reasons, is often recognized only with difficulty, but the latter two may be detected early by the employment of laboratory measures. In a word, beware of latent constitutional disease.

Only recently a judge in the State of New York rendered a verdict against an insurance company's physician because he had failed to examine the urine of a patient suffering from a crushing injury to a finger with secondary infection. The surgeon operated upon the finger unaware that the patient had diabetes, although the patient claimed that she had told him. Gangrene and loss of the finger followed, whereupon the patient was awarded $6000.

### INJURIES WITH BACTERIAL INFECTIONS

When we consider the nature of the average plant with its myriad possibilities and probabilities of bacterial presence, it is always a safe procedure to consider every wound, no matter how trivial, as certain to become secondarily infected and, therefore, treatment towards sterilization of the wound should be immediately instituted. The comparative ease with which that may be done depends largely upon the nature of the wound and the experience of the plant surgeon. Suffice it to say that small pin-prick wounds are the most dangerous and at the same time the most neglected, often by the injured worker, but also not infrequently by the plant surgeon. Blisters, the result of an unaccustomed occupation, such as sawing logs, etc., almost always become infected because of the close proximity of contaminating material. Infection following an insect bite offers an interesting and not undisputed problem. Such injuries frequently become infected, but whether due to bacteria in the animal or bacteria from the injured person's skin is a disputed

problem. The occupation of the individual at the time of the injury frequently gives a lead as to the possible type of infection: thus tanners, workers in skins, etc., are apt to contract anthrax, whereas the worker in the fields (garden soil) frequently becomes infected with tetanus. The following is a rough classification of groups of microorganisms, which it would be well to bear in mind, since they give a clue to treatment:

1. Pus-producing bacteria: streptococci and staphylococci.
2. Gangrene-producing bacteria (putrefactive bacteria): Bacillus coli-communis, bacillus of malignant edema, Bacillus histolytica, Vibrion septique, etc., Bacillus welchii.
3. Bacillus tetani.
4. Bacillus anthracis.
5. Bacillus diphtheriae.
6. Bacillus paratyphosus or botulinus from the ingestion of contaminated food.

### INJURIES CAUSED BY BLUNT FORCE

Injuries caused by blunt force are many and varied indeed and have caused considerable discussion and litigation. An interesting article has been written on this subject by Vance, dealing with injuries to the soft parts, and one by Irving Gray on traumatic gastric ulcer. One of the frequent lesions encountered is osteomyelitis following a blunt injury. These cases always present a quiescent primary focus (paronychia, furuncle, tonsil, etc.) from which the microorganisms invade the blood stream and, following the well-known rule of *locus minoris resistentiae*, set up the inflammatory process. The microorganism encountered most frequently is the staphylococcus, although the streptococcus, pneumococcus and typhoid bacillus have also been found. The tubercle bacillus and gonococcus, as a rule, involve the epiphysis or a joint. Gelpke points out that not only crushing injury to a bone may cause osteomyelitis, but that over-exertion may cause the lesion, i.e., osteomyelitis of the sternum developing after a rowing race, or osteomyelitis of the tenth dorsal vertebra following the pushing of a heavily loaded wheelbarrow up a hill. Furthermore, he cites cases where the lesion developed following exposure to wet and cold, such as working in a water-filled ditch. However, in such cases, the onset of the osteomyelitic symptoms must occur within a definite time and not weeks after. For example, Thienes (quoted by Gelpke) showed that in 102 cases of traumatic osteomyelitis, 95 presented symptoms within eight days after onset of the injury and the majority of these within the first three days.

### JOINT DISEASES AND INJURY

There is no question about the possibility of the development of acute septic arthritis in a joint following direct injury. By far the greater number of joint injuries that come under observation are of a different nature, however. The plant surgeon must differentiate between acute rheumatic polyarthritis, arthritis deformans, gout, tuberculous, gonorrheal and even syphilitic joints. The number of cases of stiff knee joints or spondylitis deformans that clutter compensation clinics and courts is legion. European authorities give considerable weight to the question of injury aggravating a pre-existing lesion, an effort which, in the opinion of the writer, is entirely unwarranted. Such cases must be subjected to extensive roentgenological, serological and chemical study in order to eliminate the presence of organic disease. A careful history and roentgenological study will frequently show extensive old arthritic changes in many joints of the body which were present long before the alleged injury, such, for example, as hypertrophic osteoarthritic changes in individuals over thirty-five years of age which are of no clinical significance. The same applies to syphilis, tuberculosis and gonorrhea.

### BLOOD DISEASES AND INJURY

Blood diseases rarely come under consideration as the result of mechanical

injury, although a number of them are produced by industrial poisons. Of occasional importance in this connection are hemophilia, purpura (peliosis rheumatica) and hemophiliac-like bleeders. Cases of prolonged, severe bleedings and even death have been reported following trivial cuts of the skin, or blows upon the chest or abdomen with rupture of a vessel and internal hemorrhage.

Varied blood pictures, pernicious anemia-like, some secondary anemias, regenerative anemias, etc., are found in many industrial poisonings, i.e., coal gas, manufacture of chloroform, carbolic and hydrocyanic acid, in lead workers, benzine and aniline plants, and in workers in radium, thorium and *x*-ray, as well as in many other industries.

In isolated cases compensation judges in some of the European countries have allowed payments for leucemia and held that they were caused by severe general body injuries involving the spleen, bones (bone-marrow), etc.! Medical authorities believe, and the writer concurs in this belief, that thus far there is not sufficient evidence to show that any leucemia, except the leucopenia occurring in *x*-ray and radium workers, has been caused by traumatic or chemical injury.

### ARTERIOSCLEROSIS AND INJURY

Frequently claims are made that, following injury, a patient developed the symptoms of arteriosclerosis, hypertension and nephritis. These cases tax physician, counsel and judge alike and volumes have been written on the subject. It is difficult to conceive how injury can induce sclerosis of the arteries, except possibly locally at the site of injury and when accompanied by extensive suppuration and prolonged healing. The courts, however, have held, especially when the injury was to the head, followed by concussion, that it aggravated a pre-existing arteriosclerosis. In this connection, it is interesting to cite a case, the like of which has undoubtedly occurred many times.

A sixty-year-old man, under observation, fell from a chair. Thereafter vertigo, headaches, loss of memory and change in his mental behavior resulted. It was held that he was suffering from aggravation of a pre-existing arteriosclerosis. The conception of the fall having been caused by an increasing arteriosclerosis (cerebral anemia or spasm) was apparently not considered.

Peripheral arteriosclerosis, Raynaud's disease (angio-arteritis obliterans) causing gangrene, especially in the lower extremity, and trophic ulcers, etc., frequently quite innocently are believed to be caused by trauma. It is apparent that injury to such a part will cause aggravation of a pre-existing lesion or induce gangrene from destruction of diseased vessels. But here again proof of unquestionable character concerning an injury must be given, since many times injury is claimed when none occurred and the patient is suffering from a medical condition.

Patients suffering from arteriosclerosis should be carefully examined and questioned concerning their use of alcohol and tobacco, infections, and general dietary and living habits. In this connection attention must be called to the syphilitic and lead scleroses, which, however, show a different picture from that of general arteriosclerosis. It is well to remember too that advanced sclerosis in comparatively young people (thirty to forty years) is frequently caused by syphilis.

### CIRCULATORY DISEASE AND INJURY

Acute (suppurative) arteritis, aneurysms in peripheral vessels, phlebitis, pyemic phlebitis, embolism (postoperative), etc. do result from an injury, although, in cases of phlebitis, non-traumatic medical conditions, particularly infections, must never be overlooked. All statistics indicate that disease much more frequently causes phlebitis than does trauma. Traumatic aneurysms of the peripheral vessels must also be considered.

Embolism of whatever nature may follow injury. Particularly interesting is

fat embolism, occurring most often after fractures of the long bones. Fat embolism has been described in nephritis without injury, so that even in these cases a pre-existing organic lesion must not be lost sight of.

Aortic aneurysms can hardly ever be considered as of traumatic origin, particularly as one can generally prove the presence of syphilis or arteriosclerosis. However, it has been held that trauma causes aggravation of symptoms, rupture and death. Similar conditions obtain in ruptured hearts, myocarditis, angina, syphilis, etc. Recently coronary thrombosis and heart block have been invoked as compensatory injuries. An interesting case under consideration, in which a final decision has not yet been rendered in the courts, is one in which an estate claimed compensation death benefits because, after a heated argument with his employer, the employee, who had been suffering from hypertension and arteriosclerosis, dropped dead. Postmortem examination revealed coronary thrombosis. Claim is made on the basis of aggravation of pre-existing disease.

### RESPIRATORY LESIONS AND INJURY

There are many symptom-complexes referable to the respiratory tract in which injury is alleged. Some, like silicosis, are of unquestionable industrial origin, but result only after long-continued irritation from inhaling dusty air. The Schönberger lung cancer is another such example. There are cases in which the individual suddenly develops asthma, even though he has worked in the industry for years.

Thus, a baker who had worked in a bakery since his eighteenth year, at the age of forty-five developed asthmatic symptoms which were aggravated during his hours in the bakery. Careful testing showed a strongly positive reaction for wheat. He was advised to wear a respirator during his working hours, change his clothes before removing the respirator, and desensitization treatment was given. This patient recovered entirely.

European authorities hold asthma as a compensable disease. Until recently this has not been the case in this country. Today some states list asthma as an industrial disease and, therefore, compensable. In view of the fact that there is a history of heredity in almost 75 per cent of all cases of asthma, hay fever and other allergic disease and because a cure can very often be effected by change of occupation, it seems scarcely right that asthma should be listed as a true occupational disease.

Bronchitis, emphysema and bronchiectasis are frequently attributed to gassing from irritating or poisonous vapors or fumes, and it has been held in some instances that such fumes aggravated, but did not cause, pre-existing tuberculosis. Spontaneous pneumothorax, although believed by almost all lung specialists to result from a pre-existing tuberculous focus which has ruptured, is generally compensated in industrial courts.

Pleurisy from puncture by a rib after a blow has been described and considered compensable, but there is considerable dispute concerning tuberculous pleurisy in such cases and the most that can be said for it is that trauma may aggravate a tuberculous pleurisy. Pneumonia following a blow on the chest has been held compensable. Pneumonia following an injury in any part of the body is held compensable as a rule.

### DIABETES AND INJURY

True diabetes mellitus, or so-called diabetes mellitus, may follow trauma, particularly if the injury involves the pancreas or the skull at the base. It may, particularly in the latter case, be transitory, due to hemorrhage into the lateral ventricles. It is advisable to obtain the urine of a patient suffering from fracture of the skull as soon after the accident as possible, to exclude, if possible, the presence of sugar in the urine prior to the accident. Carbon monoxide and dioxide are said to have caused diabetes. Shock also is known to induce it. Diabetes

insipidus is known to follow skull injuries, particularly, it seems, those in which the cranial nerves are involved.

The frequency of diabetes independent of any cause, i.e., a purely constitutional disease, makes it necessary to exercise extreme caution before rendering a decision as to an "accident diabetes." One must bear in mind that elderly, obese, gouty and nervous individuals are predisposed to diabetes. If possible, reports on urine taken prior to the accident should be sought and a careful history taken concerning symptoms of diabetes. The fact that traumatic diabetes frequently disappears and that moderate physical exertion is of value in diabetes, should be taken into consideration in making an award. Cases of diabetes occurring several years after an accident should not be considered as of traumatic origin.

### ALCOHOLISM AND INJURY

That the finer nervous reactions are lost in patients under the influence of alcohol and that they, therefore, are a greater risk in industry is too well known to require discussion. On the other hand, in very few other conditions is there such tremendous variance with regard to individual reactions as in imbibers of alcohol. Many accidents occurring during holidays, etc., are directly traceable to acute alcoholism and in many instances in this country have not been held compensable. Delirium tremens following an injury has frequently been the first indication that a worker was an alcoholic. Alcoholism in an apparently normal individual retards healing processes at times. The question of alcoholism revolves about a careful history as to whether the injured worker is an alcoholic, the amount of alcohol imbibed and, if possible, the determination by chemical means of the presence of alcohol in his blood or spinal fluid during life, or in the viscera after death.

### SYPHILIS AND INJURY

Syphilitic infection comes under consideration in two ways. First, we may have extragenital infection in doctors, nurses, orderlies, etc., occurring in the course of their work, or in such manufacturing operations as glass blowing, or infection may occur as a result of shaving in a public barber shop, etc. Secondly, and in this country of greater importance, the tertiary and congenital lesions play a greater role, particularly in bones and joints (Charcot's joint). Crushing injuries, especially of joints and, to a less extent, of bones and soft parts, may fail to heal and cause long impairment of function, destruction or encapsulation of bone (osteoperiostitis), suppuration, etc. Frequently great difficulty is encountered in differentiating between osteomyelitis, tuberculosis and syphilis of a joint. Spontaneous fractures in syphilitic subjects, the result of gumma or diffuse syphilitic inflammation (osteomalacia luetica) must not be overlooked, since they cannot be considered as accidents, but must be regarded as a medical condition.

### TUBERCULOSIS AND INJURY

The fact that practically 100 per cent of the population, certainly of industrial centers, has a tuberculous focus (tubercle infection in lymph nodes, tonsils, etc.) in some part of the body and the fact that a great many tuberculous individuals and even some presumably non-tuberculous individuals may have the bacilli of the disease circulating in their blood, makes the question of tuberculosis produced by an injury a very difficult one. As in syphilis, the joints and bones are most frequently involved, although other structures (kidney, scrotum, lymph nodes) may become the seat of a tuberculous process, particularly after a crushing injury, even though prior to the accident the organs were entirely normal. The fact that microorganisms circulating in the blood find such injured tissue an ideal medium for growth cannot be denied. The authorities of different countries have held various opinions concerning the compensability of these lesions. Thus, in Germany full

compensation, as a rule, has been awarded. In other countries only partial payments are permitted. The question as to the percentage of compensation allowed seems to the writer to depend upon the condition of the injured worker, the type of injury and the course which it runs. We have, on the one hand, the anatomical tubercle (vaccination tubercle) occurring in anatomists, butchers, morgue keepers and those who handle animals and, on the other, the development of a sinus following trauma to an already swollen knee. The former certainly is a traumatic infection, the latter at most an aggravation of a pre-existing disease. Between these two extremes there are all manner and type of lesions which require most painstaking observation and careful consideration before an opinion is filed and an award given.

### INJURY AND THE ENDOCRINE ORGANS

Because of the paucity of our knowledge concerning the physiology of the endocrine glands and the pathogenesis of diseases connected with them, it is almost impossible to give accurate opinions and decisions concerning them. Thus we know from experimental, as well as clinical observation, that goiters develop from lack of iodine in the system. We also know that hyperthyroidism develops in nervous (neuropathic) individuals. Frequently shock will produce hyperthyroidism, for example this was amply exemplified during the World War. Numbers of soldiers exposed to the vicissitudes of trench life were invalided with many or all of the symptoms of Graves' disease. Can we then say that psychic or physical shock in the course of his occupation caused a worker's Graves' disease? Trauma to the skull with supposed injury to the pituitary, resulting in changes in the developmental and psychic personalities of an individual, have been described (acromegaly, gigantism, leontiasis, precocious sexual development, etc.). To severe blows in the adrenals and ovarian regions have been ascribed a whole train of symptoms, even the development of malignant growths. It is possible, but only rarely, for a severe blow to bring on spontaneous abortion, although claims for injury in connection with abortion range all the way from shock (fright) to severe crushing injuries of the abdomen or pelvis. Amenorrhea, sterility and dysmenorrhea have been made the basis of claims for injury. Of these, the last named may be the result of traumatic displacement of the uterus. Crushing injuries to the testicle are said to have produced changes in the male characteristics of a worker. That the latter can undoubtedly ensue no one will gainsay, but there are several factors to take under consideration, such as the state of physical development of the patient prior to the injury, the age of the patient, and the extent of the injury. In this connection it is important to call attention to the profound alterations in the endocrine and vegetative nervous system resulting from poisoning by thallium preparations, so often used as a depilatory. Osteomalacia, sometimes with spontaneous fractures, have been ascribed to endocrine trauma. Sudden death in status lymphaticus following a trivial injury is well known and is attributed to changes in the thymus and other structures, although its actual cause is not known. Status lymphaticus is generally not recognized until death results and, although the anatomical earmarks of the condition have been well described by Norris, Symmers and others, it is frequently overlooked even at the postmortem table. The writer has time and again noted great amazement at a spontaneous rupture of a hypoplastic cerebral vessel in a young and apparently healthy individual who showed all the signs of status lymphaticus.

Each case of alleged endocrine injury must be carefully scrutinized and studied and opinions rendered only upon most minute evidence.

### THE NERVOUS SYSTEM AND INJURY

Injuries to the nervous system also belong to one of the most difficult and

controversial chapters in medicine. I do not refer to compression myelitis from a vertebral fracture, nor to paralysis following the severance of a nerve; such cases are *prima facie* evidence of injury. But myelitis appearing suddenly in an individual without known cause (injury, fracture, compression) and subsequent claims made therefor are difficult problems to solve. The World War showed fairly conclusively that emotional disturbances (shell shock, hysterical paralysis, etc.) which continued beyond a few days occurred only in those predisposed to nervous disturbance, either inherited or acquired before the accident. Inheritance (epilepsy, migraine, personality characteristics, the psyche, etc.) unquestionably plays a greater role in the nervous or mental disturbance of patients than such external factors as worry, death, overexertion, occupation, fatigue, loss of previous possessions or comforts, on the one hand, or over-indulgence and indolence, on the other. Neuroses in general can undoubtedly be ascribed only to persons with an inherited or acquired (vascular, as, for example, cerebral arteriosclerosis) predisposition to a neuropathic state. On the other hand, such a patient often is the most difficult to deal with since he is the last to believe or reveal that there may be anything wrong with his nervous system. As regards occupation, it is obvious that an artist or musician has a more highly developed nervous system than a farm hand, for example, so that the designations, nervous constitution, sensitive nerves, etc., have some basis in fact, and occupation plays some role in the evaluation of lesions of the nervous system. The structure of the nervous system, particularly the richly vascular brain with the vessels lying in a rather soft matrix, predisposes to lacerations and hemorrhage. It is, therefore, quite natural that almost any symptoms of brain disease may develop. On the other hand, it has been pointed out that hundreds of individuals receive an injury to the skull, but only the exceptional patient develops epilepsy. Again cases have been recorded where, following a traumatic injury to the head, epileptic seizures developed, only to disappear after an operation. Traumatic epilepsy is well known. Not so, however, with such conditions as paralysis agitans, syringomyelia, multiple sclerosis, mental disease, etc. Here again each case must be decided on its individual merits and as a general rule predisposition or latent disease plays a major role. The physician who deals with such cases and renders decisions on them would do well to familiarize himself with the vast literature which has accumulated in this particular field. Attention must also be called to the fact that in no other group of diseases are the factors of simulation, exaggeration and self-inflicted injury so important, and the physician must acquaint himself with the manner and methods employed for detecting them. It is perhaps well to bear in mind the admonition of a famous neurologist of a decade ago, who said that hysteria may simulate any symptom or disease, except albumin in the urine; he had never observed or heard of hysterical albuminuria.

## NEOPLASMS AND INJURY

To attribute a new growth to the result of an injury is fraught with a great deal of difficulty for many reasons. If we pause to consider for a moment the various theories concerning the pathogenesis of tumors we immediately realize the difficulties with which we are confronted. We have, on the one hand, the theory of misplaced embryonal rests and, on the other, the theory of infection. Throughout all the theories runs the concept that "something" is wrong with the organism. In other words, we have "something" (a predisposition) in the body which, plus an injury, starts the growth; hence it is not a pure accident. Again we have the division of benign and malignant growths. In the latter group, there is a vast difference in the developmental history of carcinoma and sarcoma. Trauma as the cause of a new growth is probably attributed to every growing neoplasm, since the patient undoubtedly

had received a blow at one time or another in the region of the growth. It is generally conceded that benign growths (with the possible exception of a fibroma developing from a hematoma at the site of injury) are not caused by trauma. In malignant growths the problem is somewhat different. Of the many cases of carcinoma which a physician sees annually, few as a rule can be ascribed to injury. Nevertheless, the paraffin cancers, tar and chimney sweep cancers, Schönberger lung cancer and "aniline" cancers of the bladder are too well known to require further discussion, although attention must be called to the particular type of injury in these cases, i.e., a protracted irritative trauma which is believed to prepare the tissue for the tumor implant. Whether or not a crushing injury to a part may produce carcinoma is a moot question. Sarcoma developing in a callus is by no means a rare finding. The occurrence of sarcoma in connective tissue following a blow or injury is also recognized. The development of sarcoma in a radium osteitis is also known. However, cases have been recorded in which spontaneous fractures occurred as the result of tumor growths in the bones without a history of injury. Keloids, although a form of tumor from mechanical injury, may hardly be considered a new growth.

In estimating injury as the cause of a tumor growth, we must consider the following:

1. The predisposition of the individual to a new growth (age, occupation, nature of the injury).
2. An unquestionable accident must have taken place.
3. There must be no signs that the growth was present prior to the accident (no metastases, no spontaneous fracture in a bony growth).
4. The growth must occur at the site of injury.
5. The growth must occur within a given time (sarcoma soon after an accident, carcinoma as a rule after a longer period of time).

From these factors one may conclued that carcinoma resulting from injury is a rare finding. Sarcoma, principally from bone callus, etc., less often from soft parts, is of more frequent occurrence.

Aggravation of an existing new growth by injury, particularly crushing injury, is generally conceded, though in the case of solid tumors experimental evidence is not conclusive. Cystic tumors, however, may readily be injured by trauma. Thus ovarian cysts have been known to become twisted as a result of trauma or ruptured from severe blows, stab or bullet wounds, etc.

## SUMMARY

In this paper no attempt has been made to describe in any detail the various organic lesions which may be created or influenced by trauma, but merely to point out a few of the more important groups of organic disease which come under discussion. The reader must search the rapidly accumulating literature of industrial medicine, accidents and public health in order to familiarize himself with the various details of a given case. Fortunately monographs on special subjects are constantly making their appearance. The writer has not made a systematic search of the literature, but has appended a list of books and journals which will be of value for reference.

Much of an academic nature has been omitted from this paper. Thus the question may well be raised as to what constitutes trauma or injury. Legal opinions differ on the definition, though it appears to the writer that any chemical, biological, physical (including thermal) or mechanical agent which, when introduced or applied to the body causes dissolution of the normal physiological processes in the individual, must be regarded as the cause of injury. However, when an individual cuts himself in his own home, or develops poisoning as a result of continuously drinking out of a vessel containing antimony, arsenic or lead, the case is not one resulting from industrial accident and, therefore, is not medicolegal. If, on the

other hand, he cuts himself with the same knife in his place of employment, or becomes poisoned by lead because he handles the metal in his occupation, the case immediately assumes medicolegal aspects.

Attention must again be called to the constantly recurring question of simulation or exaggeration of trauma and the self infliction of injuries for the purpose of collecting compensation. Such cases are far more numerous than was anticipated by the framers of our accident and compensation laws and are frequently decided in favor of the complainant by careless and not too scrupulous referees. To the warning, beware of latent constitutional or pre-existing disease, may be added, beware also of simulation, exaggeration and self-inflicted injuries.

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# ROENTGENOGRAMS AS A MEANS OF IDENTIFICATION

FREDERICK M. LAW, M.D.

NEW YORK CITY

IN 1927 I reported with Dr. William L. Culbert[1] a case in which the existence of roentgenograms of the sinuses and mastoids made in 1918, made possible the identification of the body of a man who in 1925 died under such conditions that the ordinary means of identification of his remains were of no avail. A considerable estate was involved in the proof of death of this individual, and the identification was sufficiently convincing that it was accepted in a court of law. The details of the case were melodramatic beyond the imagination of the writers of the most lurid of mystery tales. The person in question disappeared from a camp on the Indus River in Kashmir; seventeen days later the bodies of two white men were found in the river, 70 miles below camp, dismembered and disfigured beyond recognition. Both bodies bore the scar of a left-sided mastoid operation, one a radical and the other a simple operation. The scar of the simple mastoid operation constituted presumptive identification of the remains as those of the person whose roentgenograms were in our possession; and after considerable exchange of correspondence between the U. S. Department of State and the Indian government, the latter waived its usual rule of forbidding a body to be shipped out of the country uncremated. The remains arrived in a hermetically sealed container via freight steamer from India, and were delivered at the New York City Morgue on February 23, 1926, 260 days after the individual had disappeared from his camp.

Fingerprints on record would have been of no aid, for one arm was gone and there was scarcely any flesh on the bones of the other hand. The facial bones were almost bare of flesh. However, an examination of the sinuses and mastoids with a portable machine at the morgue, and comparison of the resulting films with the plates on file, established beyond any possibility of doubt that both sets of roentgenograms were of one and the same individual. The operation on the left mastoid following the first examination had destroyed its value for identification, but as both sides had been examined, the right mastoid was available for comparison. Thirteen points of identity in the sinuses and seven in the right mastoid were noted; the number could have been extended indefinitely.

After the identity of the individual had been established by roentgen means, there was further corroboration by Dr. A. R. Starr, Jr., who as the patient's dentist had placed three gold inlays in 1924–25 which he was able to identify in the mouth of the skull.

In a survey of the literature of roentgen identification, I was not able to find a record of a case antedating this one, though Schüller[2] had suggested in 1921 the possibility of identification from roentgenograms of the frontal sinuses. Schüller's article was the only reference found on this subject.

While the sinuses and mastoids naturally furnish the most satisfactory means of identification because of their intricate structure and infinite number of points for comparison, I have no doubt that roentgenograms of other parts of the body could establish identity also; particularly the lumbosacral region, which is subject to great individual variation. Thus a torso recovered from a fire or from any of the

[1] Culbert, W. L., and Law, F. M. Identification by comparison of roentgenograms. *J. A. M. A.*, 88: 1634–1636, 1927.

[2] Schüller, A. Das Röntgenogram der Stirnhöhle: Ein Hilfsmittel für die Identitätsbestimmung von Schädeln. *Monatschr. f. Obrenh.*, 55: 1617–1620, 1921.

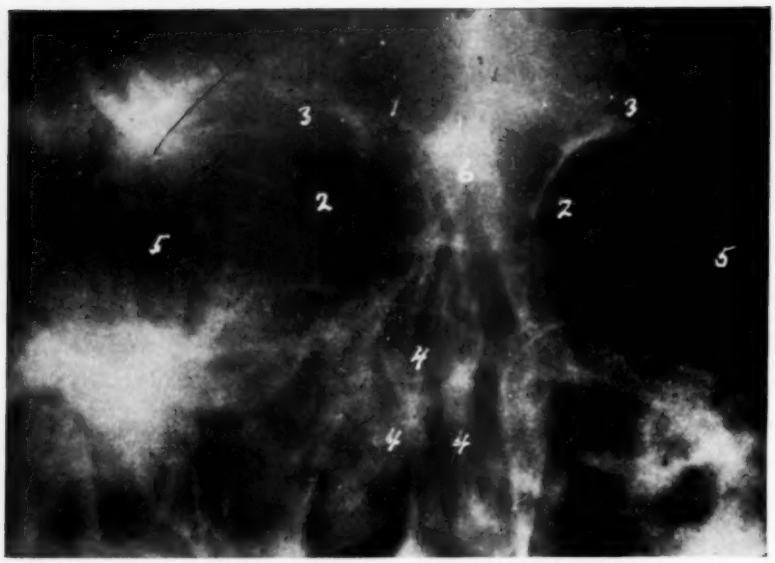


FIG. 1. Radiograph of sinuses made during life.

FIG. 2. Print of post-mortem radiograph of sinuses.

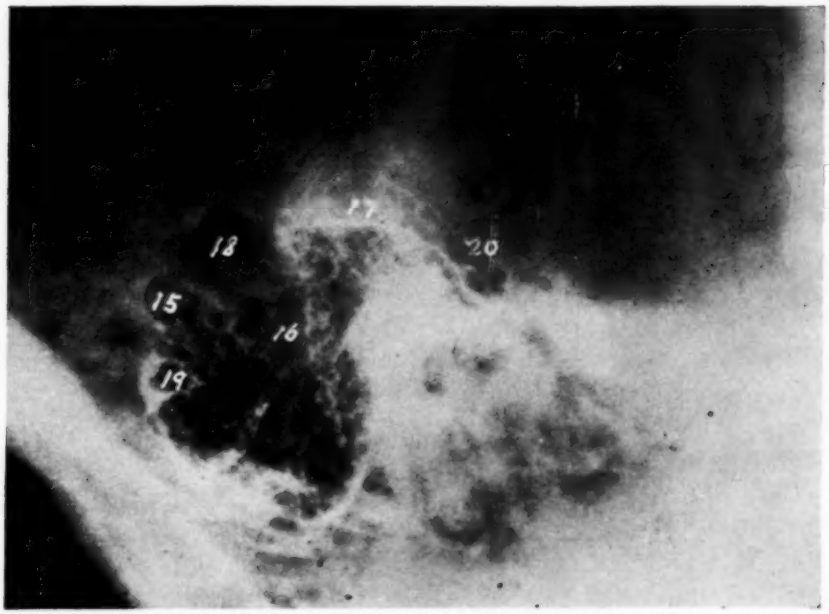


FIG. 3. Radiograph of right mastoid made during life.

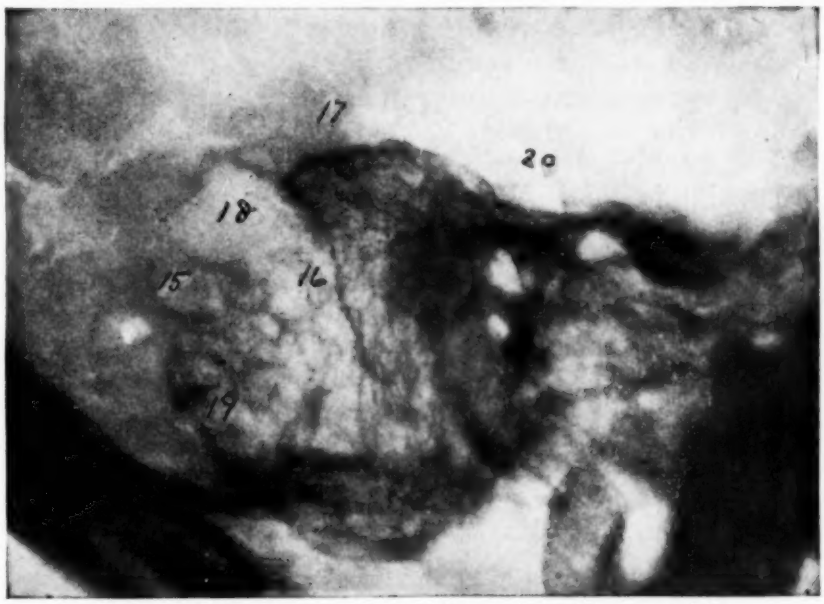


FIG. 4. Print of post-mortem radiograph of right mastoid.

situations so relished by readers of murder mysteries, could serve to establish identity if a roentgenographic examination had been made in life and the films were available for comparison.

The deliberate employment of roentgenography of the sinuses for future reference for the purpose of identification depends only on economic considerations; the feasibility of the method and validity of the identification have been fully established. While the mastoids alone will establish identity, the possibility of a bilateral mastoidectomy destroying the evidence makes the sinuses of more permanent value for future reference for this purpose; it would involve considerable extra expense to examine both the sinuses and the mastoids as a routine for military or insurance purposes, or in any situation in which the method might be adopted.

The increasing cost of storage space in private practice and the competition with other departments for the storage space in odd corners of hospitals makes it more and more difficult to maintain files of roentgenograms indefinitely. The value of such records, however, for future reference on clinical grounds alone fully justifies the preservation indefinitely of all roentgenograms except those made on the now obsolete nitrate-base film. During the transition period from the highly combustible nitrate-base film to the present acetate-base film, which the fire underwriters rate as of no greater hazard than a corresponding amount of paper (and paper filed the way these films are certainly represents very little fire hazard) there was considerable apprehension as to the fire hazard of the new films, which was founded purely on lack of knowledge of the facts. This misconception, coupled with a lack of appreciation of the subsequent clinical value of roentgenograms, led one hospital superintendent to write an article in *The Modern Hospital*, stating that such films were of no value after six months and that the fire hazard demanded that they be destroyed. Every argument in this article was promptly challenged by Dr. Ramsay Spillman,[3] the validity of whose refutation was acknowledged in a personal letter from the author in question.

It would doubtless be difficult to persuade hospital superintendents to allot storage space for the indefinite preservation of roentgenograms on the grounds that one case out of say ten thousand might serve to establish the identity of a person in a medicolegal case. However, the clinical value of roentgenograms for reference at any future date, however remote, should bring about the indefinite preservation of such films, and make them available for such medicolegal need as may arise.[4]

It goes without saying that for films to be of value for future reference for identification of a person, the films should be identified with a marker which is radiographed onto the film. Adhesive labels in the course of time fall off, and confusion could be introduced into a case when the opposing counsel could raise the question of doubt as to the identity of the original films in a case of the nature of the one described here.

[3] Spillman, R. Why *x*-ray films should be preserved indefinitely. *Modern Hosp.*, 35: 63, 1930.

[4] While this article was in the compositor's hands, Dr. Spillman saw a case in which a claim for added disability benefit was awarded by a life insurance company because the claimant's physician was able to submit a film of the chest made in 1928, showing advanced tuberculosis antedating the beginning of the payments.

# SUGGESTIONS FOR IMPROVING MEDICOLEGAL COURT PROCEDURE

RAMSAY SPILLMAN, M.D.

NEW YORK CITY

THE late Dr. George W. Gay[1] of Boston stated in 1909:

> Some of our most accomplished experts utterly refuse to appear in court or to accept service as an expert under the present methods of conducting examinations. They very naturally object to placing themselves in a position in which they may be subjected to the taunts, to the ridicule and to the insults of third-rate lawyers. They are gentlemen endeavoring to serve the cause of justice to the best of their ability, and hence will not submit to the humiliation of being treated as adventurers and mountebanks.

In the same journal for January 6, 1916, is published an address by Dr. J. W. Courtney of Boston, to the Harvard graduating class of 1915:

> It is the bounden duty of every man in the profession so to shape his conduct toward cases which promise to eventuate in court proceedings, that due respect will be given his opinion, that he will not merit the biting sarcasm, the sneers, the raillery and general browbeating of opposing counsel.

At a hearing before a special meeting of the New York County Medical Society in May, 1934, a very distinguished attorney spoke of having defended a physician "in the face of the leers and jeers and sneers of opposing counsel."

Here is evidence covering a period of twenty-five years, and the latest evidence is from a lawyer and not from a doctor, that sarcasm, raillery, brow-beating, leers, jeers, and sneers are a part of the sanctioned legal procedure today; and so long as that is true, so far as I am concerned, law is the problem-child of the learned professions, to be treated as such. If the legal profession desires to be treated as an equal by the other learned professions, I should recommend that it outgrow its tantrums in the courtroom and act its age. Until it adopts very different tactics on several points in its procedure, I can answer for one roentgenologist who will cast his lot with the experts mentioned by Dr. Gay twenty-five years ago; as for the standing of Dr. Gay, the record can show that at the time he made the statement cited, he was a past president of the Massachusetts Medical Society.

Based entirely on my own observation of the legal procedure as I have seen it myself in court, I have formulated a number of reasons for stating before the New York Society of Medical Jurisprudence on May 14, 1934, that so long as I am a free agent I shall never again appear in court as an expert witness. On the evening mentioned I went into considerable detail in the exposition of those reasons, and the legal members present varied in their reactions from amused tolerance to apparently grave danger of apoplexy. In fact, it was the liveliest meeting that the society had had since the night that Clarence Darrow had been advertised to appear and did not show up.

I can't say that any lawyer ever tried to abuse me in court, so the browbeating tactics of opposing counsel constitute only a minor reason for my own personal distaste for court work. However, I have said before and I reiterate it here, that a legal procedure which countenances an opposing counsel resorting to leers and jeers and sneers is thoroughly vicious; it has no legitimate place in human affairs; and so long as this is true of legal procedure I shall never be its voluntary hired man

[1] *Boston M. & S. J.*, 161: 969, 1909.

again. This should not be construed as my stating that I hold the legal profession in contempt. I am proud to claim the friendship of not a few distinguished lawyers who are throwing their influence on the side of purging the legal procedure of these tactics.

I shall briefly recapitulate here my reasons for distaste for participation in present-day medicolegal procedure in negligence cases, as given in the paper read before the Society of Medical Jurisprudence:

1. It seems to me that when two people go to law, justice is on the side of only one. Therefore the expert witness runs at least an even chance of selling his services to the side of injustice.

2. An expert goes on the stand and is sworn to tell the truth, the whole truth, and nothing but the truth. However, I have repeatedly observed in court that attorneys in the very nature of their job under present procedure, are no more interested in bringing out the truth as it helps their side, than they are in suppressing the truth as it is prejudicial to their side. It is possible, therefore, for the expert under present procedure to tell the truth and nothing but the truth, but in view of the right of the opposing (or even his own) counsel to object, it is practically impossible to tell the whole truth. This statement has the support of an utterance of Dr. Gay (*loc. cit.*): "As the writer understands the matter, it is a very small part of the duty of the counsel in any suit at law to see that justice is done. It is their duty to look after the interests of their clients."

3. In the reference cited, Dr. Courtney stated in 1915:

> The present mode of procedure in our courts is, so far as medical testimony is concerned, not a particularly edifying one. To illustrate this point, let us take, for example, a case of the type which is most commonly met with in every day work of the courts—an action of tort for personal injuries. In such a case, the plaintiff is practically always of the proletariat class; the defendent, a public service corporation, or an insurance company. The array of witnesses on either side is generally appalling. Of these the medical ones alone concern us. They are of two hostile camps and prepared to attempt under solemn oath, to uphold opinions diametrically opposed, yet supposedly derived from a single series of facts and observations.

Therefore: when justice would demand judgment arrived at by calm deliberation on the part of experts who know what it is all about and who can weigh the pros and cons and finally come to a decision, the final opinion is left to twelve citizens who know nothing of medicine, and who could scarcely give a valid scientific opinion even if the only appeal made were to their reason. When on top of it all it is considered that lawyers on two sides have wooed[2] the emotions of this jury with all the arts and wiles that are summed up under the term "forensics," when it is remembered that a wife with a nursing baby in court is worth to a plaintiff more than two medical expert witnesses (which is my opinion based on my own observations, and you are at liberty to disagree with it if you like), so far as I am concerned, medicolegal procedure in court today is often nothing but burlesque; and while burlesque seems to have a place in the great social scheme, I hold that medical science has no place in burlesque.

I might mention in passing that lawyers have made much of the inability of doctors to agree. It is my observation that disagreement between doctors is more common in court than it is in the hospital. I have been a part of many *x*-ray conferences where doctors would come into the case with divergent opinions; but as added evidence of one kind or another was introduced and the case was discussed dispassionately and scientifically and the added evidence was weighed pro and con, as a rule there would develop some degree of unanimity of opinion; this is as it should

[2] Assuming that both camps of counsel have exercised all the prerogatives that accepted procedure sanctions.

be, for as Dr. Courtney stated in 1915, these opinions are derived from a single series of facts and observations. But the court procedure of today could not be more unfavorable to a scientific study of a case by experts if it had been devised with malice aforethought.

4. The legal profession has tyrannized over the medical profession with no mercy at all, on the matter of subpoenas. As a would-be law-abiding citizen, I should have the constitutional right to pursue my legitimate pursuits where and when I please. If I make an appointment to see a patient in my office on Wednesday morning, and the patient is postponing other business to see me at that time, I should have the right to feel confident that barring the ordinary hazards of life in a big city, I can see that patient. But any lawyer, be he ornament of the bar or just plain bar-fly, can send a process-server into my office on Tuesday just before I leave for home, perhaps after my secretary has gone, and if I open the door myself I may find a subpoena in my hand commanding me to lay all other business aside and appear at such and such a court at 10 A.M. Wednesday; unless I go, I risk a term in jail for contempt of court. The case is more likely than not to be some fracture case of which I saw the films at the hospital three years ago, and pro-rating my monthly honorarium at the hospital for that case my original stipend for my services to that patient may have been some such figure as eighteen cents. I am assured that if I am put on the stand as an expert I will be paid as an expert. How do I know in advance whether I will find myself appearing as an expert the following day for a respectable crowd, or for a gang with whom I would not be found dead at a dog-fight? The general appearance of the process-server is more often than not a promise that the latter will be the case. I deeply resent the indiscriminate and unintelligent use of the power of subpoena; I can assure the legal profession that a great many other doctors do also; and I advise the latter not to remain inarticulate on the subject if they do not like the present situation.

The criticism of the legal profession by a distinguished physician nearly twenty centuries ago that is recorded in Luke XI, 45 et seq., finds a cordial echo in the heart of many a medical man today. The situation of which the present paper is a complaint is therefore not a new development. However, there was probably never a time when more concentrated effort was being exerted on the legal profession by public opinion than now; and it is encouraging to note that there seems to be just a little daylight ahead. I wish the record to show that the entire contents of the paper which was so little to the liking of some lawyer members of the Society of Medical Jurisprudence have been endorsed *in toto* by Dr. R. H. Kennedy, who is surgical director of the Beekman Street Hospital, by Dr. Walter L. Niles, Vice-President and trustee of the New York Academy of Medicine and by a number of other representative physicians and surgeons. Physicians who share my own desire for several radical changes in medicolegal procedure in action of tort for personal injury, will be interested in a letter addressed by Dr. F. E. Elliott of Brooklyn, New York, to Dean Charles K. Burdick, chairman of the Law Revision Commission for New York, outlining the shortcomings of the present procedure and suggesting changes for its improvement. That this document is to receive consideration is indicated by Dean Burdick's courteously acknowledging it with a request for additional copies for the other members of the commission. With the kind permission of both Dr. Elliott and Dean Burdick I append the letter, dated July 9, 1934, and commend the support of the medical profession for its fulfillment, by whatever means they have for making their opinions known. While the letter in question deals with New York state, so far as I know the situation is not greatly better anywhere else in this country, and the example could well be emulated in other states.

July 9, 1934.

Professor Charles Burdick, Chairman,
Law Revision Commission,
Cornell Law School,
Ithaca, New York.

Dear Sir,

If it comes within the scope of the study of the Law Revision Commission may the people of the State hope for recommendation of very considerable change in the rules of legal procedure in the so-called personal injury "negligence" claim.

From frequent court experience, as medical expert witness during the past sixteen years, these criticisms seem well founded and constructive.

1. It is unreasonable to assume that the memory of witnesses can retain definite and precise details of events two to seven years after the incident upon which an alleged cause or claim is founded. The "refreshing" of the memory by legal counsel is a travesty on Justice: a transparent legal fiction to cover "coaching" of the witness. By artful "suggestion" many witnesses are induced to unwitting acts of perjury: actually, the trial is too often a tournament of liars with lawyers calling the signals. The lapse of time, caused by delay of trial, favors the unscrupulous and therein tends to defeat justice. The determination of "negligence" and its implication, "liability," should be made a matter of record without delay and while the facts are still fresh in the minds of the concerned witnesses.

2. The facts and arguments of alleged negligence are ordinarily within the range of ordinary lay-mind experience. There are exceptions to this generalization: some plan for a technical jury would serve the ends of justice in these special instances. Certainly, the lay jury has served to the maximum of its intelligence-quotient when it has settled the basic question of whether or not there has been personal injury arising out of negligence, modified or not by contributory negligence: the fact "liability" or "non-liability."

3. The evaluation of medical testimony is certainly beyond the average jury-intelligence.[3] Expert medical opinion lies beyond the full comprehension of even the attorneys, with that unusual exception in which the attorney of one side or the other has properly prepared himself for some particular problem trial.

4. The medical expert should be the servant of the Court: so employed and compensated and entirely independent of any interest in the case at issue. He should be subject to interrogation only by or through the Court: never directly by the partisan attorney, the purpose of whose question is too often a theatric appeal to prejudice or emotion or other frailty of jury mentality.

5. Medical witnesses are subjected to two efforts at distortion of their testimony. One attorney urges an over-statement of medical opinion to favor his client, and the other tries to discredit and destroy the truth of the testimony. The presentation of medical testimony is commonly a mockery on science: a drama staged for its influence on the emotions rather than a procedure for the exposition of Truth.

6. The settlement of a claim for personal injury is therefore a three stage process. First, the determination of the question of "liability." Second, determination of the character of injury: and its resultant effect upon the health and functions of the injured: and the degree of permanency of effect. And third, determination of the amount of reward, if any, due the injured from the party guilty of the negligence-cause.

7. The report of the Economics Committee of the Medical Society of the State of New York, February 15, 1933, page 3, Sec. 18, Subdivision 4, dealing with problems of injury under the Workmen's Compensation Law, suggests an approach to a more sensible appraisal and determination of degree and consequence of injury. A disinterested opinion reduced to tangible terms of percent of function should be the basis for awards rather than the worked-up sentiment and emotion of a jury of laymen.

8. Justice Frederick Crane has suggested the hearing of these claims by a commission composed of three, one physician, one layman and one attorney. Such a plan appeals to reason: because of its simplicity; because settlement would be prompt and with less burden upon the community both in point of time of witnesses and of taxpayers' money: "Justice delayed is justice denied."

[3] "Jury-experience" conveys the meaning on this point better than "jury-intelligence," in my opinion. A jury can be highly intelligent, and still for lack of medical background be incompetent to pass on the value of medical expert testimony. R. S.

9. "Contingent fees" should be prohibited: they stand in the way of "settlements out of court," and not uncommonly an award to the attorney of extravagant compensation for a trifling service. The jury or commission which fixes the award to the injured should also fix the fee for the attorney representing the plaintiff. This will not appeal, of course, to the legal profession: however, some new scheme of determination of legal compensation should be found before the lay masses of our land sovietize American justice and treat the profession of Law as they did in the recent Russian civil turn-over. The citizen who has suffered a crippling and permanent injury and who sees half of the award for that injury go to the attorney has had a profound lesson in communism. The unrest of the literate mass is a matter for serious consideration by all professional minds. This settlement of claims for personal injury is one important item in a long list.

10. Casualties of non-industrial life, arising out of alleged negligence of another, occur in such increasing numbers, that an awakened and growing public sentiment must shortly insist upon some more satisfactory machinery for settlement. Some awards are absurdly small and some unreasonably large: and practically all are too long delayed. The process of settlement is too costly in wasted time of citizens, called as witnesses, and in money to both sides of the litigation.

11. The professions of Law and Medicine must cooperate to amend this unhealthy social fault, or suffer further loss of prestige or more serious consequence. If I sense the temper of the man-in-the-street, a policy of "do nothing and wait" leads to a day of reaction in which our intelligence may have little influence: we may better plan now while it remains within our prerogative.

Respectfully,
Frederic E. Elliott, M.D.

# MEDICAL TESTIMONY

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NEW YORK CITY

THE relationship between injuries suffered through accident and disease claimed to have been aggravated or accelerated by such accident has always been the subject of litigation. For this reason a lawyer must be somewhat familiar with medical questions, and is always interested in the manner in which the doctor or surgeon presents his testimony from the witness stand. In past years cases in which damages were sought for personal injuries were tried before a jury. In recent years and since the enactment of the Workmen's Compensation Laws, the great majority of cases which arise out of industrial accidents are tried before a referee or other judicial or semi-judicial officer. Jurors have little knowledge of medical questions, but in the administration of the Workmen's Compensation Law, where medical testimony is given daily, an entirely different situation is presented. The referee or the judge who continually handles such cases becomes familiar with the relationship between accidental injuries and diseases. This makes it important for the doctor or the surgeon who acts as a witness to be sure of his subject, and to have sound grounds for any opinion which may be expressed by him. I am making a few observations as a result of my experience covering the past seventeen years.

We have two classes of physicians who give testimony and representatives of both classes appear very frequently. One is the general practicing physician, and the other is the physician or surgeon who specializes in some branch of medicine or surgery. The latter is commonly referred to as an expert witness, but it is wrong to say that the general practicing physician is not also an expert. The difference between the two lies in the fact that the specialist has had a much wider experience in his particular branch of medicine, has a more complete knowledge of the question involved, and for these reasons his testimony is entitled to greater weight. In the final analysis the weight of the testimony is the only real distinction between the testimony of the expert and that of any general practicing physician. It sometimes happens that the same witness presents himself as a specialist or expert on so many branches of medicine that the weight of his evidence is weakened.

The physician or the surgeon who has had actual treatment of the particular case is of course at a great advantage, and many times his testimony will outweight the opinion of an expert who never saw the case and bases his opinion upon assumed facts in the form of a hypothetical question. The surgeon who performs an operation has a great advantage because he has the actual knowledge of what he has seen with his eyes and felt with his hands. The same is true of a person who performs an autopsy, although cases are presented in which it seems apparent that the autopsy was not complete enough for a diagnosis. Recently a prominent surgeon performed a hernia operation, which was followed in a few days by sudden death. The autopsy revealed congestion of the viscera, which was given as the cause of death, whereas the surgeon believed that the man died from a pulmonary embolism following the operation, and also believed that the autopsy did not go far enough to reveal this fact.

The weight to be given to the testimony of a physician in any given case depends on many considerations. The history, both before and after the accident, and the personal knowledge of the physician, are of utmost importance. In a recent case a man was found on the street in a dazed condition, apparently suffering from some form of epileptic seizure. Nine months later he died of a tumor of the brain. The point involved was whether he fell and received a head injury which either caused or aggravated an existing tumor. The case turned upon two factors, the first of

which was the history subsequent to the accident indicating that he returned to work within a day or two, and made no complaint and had no symptoms for several months thereafter. The other was the knowledge of the operating surgeon who found no injury to the tumor or surrounding tissues. The experience of the witness in similar cases adds much strength to his testimony; also his knowledge of similar cases reported in medical literature.

The importance of knowing the subject, and of research through the literature cannot be overestimated, especially where the point involved is the connection between an accident and some unusual disease. Unless the doctor knows his subject his testimony may ultimately prove worthless. This is especially true in cases of occupational diseases such as lead poisoning. Most compensation laws in their original form had no application to diseases which resulted from long exposure to some poisonous substance. Since the law has been extended occupational diseases have been given much more study by physicians and surgeons, and to carry weight, the doctor who testifies in relation to lead poisoning or any other form of occupational disease must keep abreast of the literature on the subject. A recent case involving heat prostration and alcoholism is an apt illustration of the futility of giving evidence without full knowledge of the subject.

A man collapsed at the end of a hard day's work, with the temperature and humidity very high and made still higher by the nature of his occupation. He died within an hour, and upon an autopsy the cause of death was given as heat prostration. Later there was a chemical analysis which reported 2 plus alcohol in the brain. The doctor who performed the autopsy was called as a witness and testified emphatically that death was due to alcoholism. This doctor probably did not know that 1 plus alcohol is only a trace; that 2 plus may come from a few glasses of beer, leaves no condition which can be noticed by others, and means little, if anything, in connection with sudden death.

More exaggerated cases are those in which physicians intending to be helpful appear as witnesses and testify that death was contributed to by an accident, when in some cases they do not know the cause of death, and in others they are unable to describe the accident in question. It hardly seems possible that such cases would arise, but the writer has met both classes in his own experience.

Experts usually testify upon hypothetical questions. The weight of such expert testimony depends of course on the facts set forth in the question, and upon the ability of the expert to give convincing evidence. The attorney who frames the hypothetical question must be fair with the doctor, and must include all material facts, and nothing else, otherwise the attorney, instead of his witness, will be responsible for the failure to convince the person called upon to decide the issue. At the same time the doctor who answers a hypothetical question must stick to the facts. He may draw on his own experience, and may take into consideration literature on the subject, but, as frequently happens, he must not assume facts which are not stated in the question.

In the final analysis, a doctor who gives evidence in a legal proceeding must stand or fall by his ability to convince the judge or referee that his conclusion in the case is correct. Lengthy discussions, coupled with the use of technical medical terms, detract instead of adding weight to medical testimony. The doctor who is most successful as a witness is the one who can reduce his conclusions to simple language and who can explain to the judge or referee in such language the reason why there is, or is not, any connection between the accident and the disease from which the patient is suffering or which has caused his death. An illustration of what I have in mind is cases of sudden death from coronary thrombosis or coronary embolism. The fundamental cause of such deaths is a disease of the artery, but what appeals to the trier of facts is the terminal condition which may be described as mechanical. The artery may be compared to a small rubber tube, and the passage becomes

gradually narrowed by disease. In the final state the passage becomes so narrow that a blood clot forms spontaneously. This is a thrombosis which shuts off the supply of blood entirely, and is the terminal or mechanical cause of the sudden death. The same thing happens with embolism except that in addition to the blood clot there is some foreign particle which helps in its formation. In most cases of sudden death from such coronary diseases, some connection is alleged between the employment and the death, usually a strain or the stress of heavy work. It is a matter of common knowledge that people die when they are at rest from coronary thrombosis or coronary embolism, and when the terminal and the mechanical cause is explained, it then becomes easier for the medical witness to explain how some alleged accident contributed to the final process, or convince the judge or referee that it had no relationship thereto.

In the administration of workmen's compensation laws, attempts are made to connect almost every form of disease with industrial accidents. In connection with medical testimony in such cases, I think it important to emphasize again the fact that referees and judges who hear this class of cases become quite familiar with certain recognized standards of what we term "causal relationship" between disease and accident. A doctor who goes beyond the accepted standard in such cases will have a hard time convincing anyone of his opinion. It is generally accepted that a severe trauma to the chest or any accident resulting in infection will activate a dormant tuberculosis. The opposite opinion on causal relationship appears to be true when the disease is already active and later responds to treatment. An entirely different opinion prevails in relation to pneumonia. It is generally accepted that this disease strikes the well and the sick in like manner, and death or recovery depends on the virulence of the type and the peculiar lung resistance or lack thereof in the particular individual. There appears to be an element of foreordination and predestination in cases of pneumonia and when a doctor asserts connection between ordinary trauma and ordinary pneumonia, he borrows considerable trouble. Another class of cases well-known in the compensation field is where death or disability is due to cancer. Certain cases, such as bone sarcoma at the site of a severe injury and skin cancer with continuity of symptoms from even a slight injury, are recognized as connected with trauma. In many other classes, such as cancer of an internal organ, it is difficult to convince of causal relationship except where there is direct damage to the cancer itself. These observations are made not as medical facts but rather as a lay opinion resulting from medical testimony in a multitude of cases. They illustrate the difficulty confronting the medical witness when he is met with a lay opinion already formed.

Extreme cases connected with rare diseases also arise on occasions. Myelogenous leucemia may be taken as an example, and the mere *ipse dixit* of a doctor does not go far in establishing connection between, for example, a simple fracture and this blood disease. Extreme cases also appear in ordinary diseases. In a recent hernia case the man had a hernia for a year before he had an automobile accident in which he received an injury to the leg. A doctor who saw him five months after the accident could not convince the referee by his mere say so that this long existing hernia was aggravated by the accident in question.

Medical witnesses often make a mistake by not admitting the possibilities on both sides of the question where relationship between trauma and disease is involved. Such admissions do not detract from their opinion in the particular case. In medical cases, as in law cases, it frequently happens that "each case is a law unto itself."

As a final word, and even repeating what has already been indicated, the object of the testimony and the thing for the witness always to keep in mind, is to convince the man who must decide the case.

# INSTITUTES OF LEGAL AND SOCIOLOGICAL MEDICINE

## A SUGGESTED PROGRAM FOR AMERICAN MEDICAL SCHOOLS

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SAN FRANCISCO, CALIF.

### INTRODUCTORY

THE medical schools of universities in the United States are designed to educate students in medicine and to give a more or less limited service to certain groups of the people. Primarily, and essentially, these schools are teaching organizations. However they also serve the State through clinics, hospitals and foundations in the solution of problems of medicine related to both individual and public health. This service feature of the medical school, when adequately developed, may be expanded more and more in directions worked out by university authorities on the one hand, and the state legislatures on the other.

### MEDICINE HAS SOCIAL AND PUBLIC ASPECTS

Medical schools are teaching students and developing medical graduates with almost all the emphasis placed on the relationship "physician-individual patent." Medical schools should also prepare students for the relationship "physician-public." Developments in certain medical schools are indicating the trend of these institutions to prepare themselves in this field. There are being and will be evolved medical institutions which in each state (or suitable area) will constitute the place to which medical students, medical graduates, or any person, or social or governmental group may turn for scientific opinion and advice whenever a social or public problem arises in relation to Medicine and allied sciences.

### AN INSTITUTE TO RELATE MEDICINE TO SOCIOLOGY AND LAW

One phase of development of any medical school which should be given most thorough consideration is the creation of a department or institute through which there might be taught medical and legal students, and through which the State might be served as regards: *the application of expert medical knowledge to the needs of the social order, law, and justice. The institute would serve to apply the organized authoritative knowledge of medicine and allied sciences to social problems and to the administration of law.*

### ONLY MEDICINE AND ALLIED SCIENCES CAN SPEAK WITH AUTHORITY IN THIS FIELD

Medicine's tardiness and Medicine's sins of omission have been in a definite way responsible for the lack of solution of many of society's problems, where Medicine and its allied sciences have been adequately prepared to give sound and efficient help. Medicine has had a responsibility which she has shirked. For example, she has been perfecting herself more and more in the diagnosis of disease and disability and in the treatment of diseases and disabilities by medicine and by surgery. But she has been neglecting to adequately train herself for, and wholeheartedly take unto herself, the treatment of illness and disability by scientific physical therapy and scientific psychotherapy. Only through Medicine might the capacities and limitations of these methods of treatment be defined. Medicine's neglect has led to the exploiting of these methods of therapy by cultists and quacks. Indeed medicine has joined with human frailty in giving birth to each and all of the psychotherapy and physiotherapy "schools" of alleged healing.

Similarly, only from authoritative scientific medical sources should come, or can come, the application and aid of the knowl-

edge of Medicine and allied sciences to the orderly running of society and the administration of law and justice. And unwillingness or hesitation on the part of Medicine and medical schools has led to the rising up of any army of self-styled chemists, pathologists, psychiatrists and "criminologists." These persons have noted the public's appreciation that *medicine is related to law and sociology* and they have answered the public demand for advice and testimony.

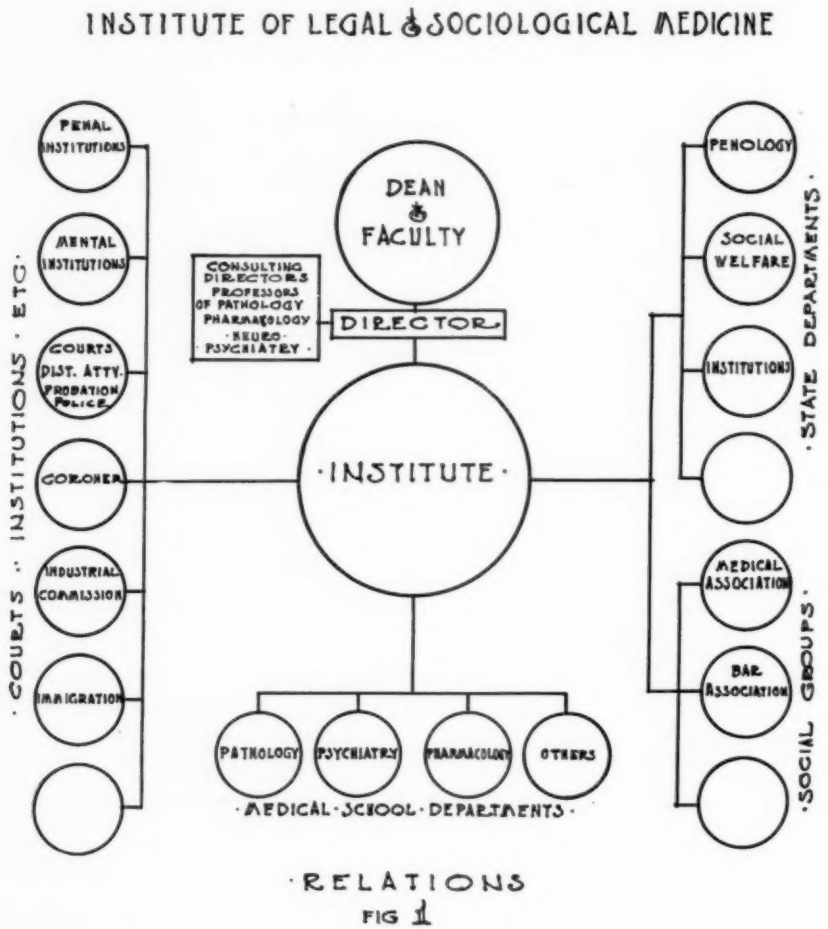

FIG 1

A violent death has occurred: The Court must know the causes. At present, in most communities in the United States the pathological and toxicological reports obtained through coroner are far short of the completeness and accuracy which are necessary to justice.

Identification of a living or of a dead person is necessary! In most communities of the United States a self-styled "criminologist" takes his scope and his materials and goes into his "laboratory" and reports glibly and verbosely concerning blood and hair and other body tissues. Surely, in the lack of adequate basic training in medicine, especially laboratory medicine and pathology, the findings of such a "criminologist" are far short of completeness and accuracy and truth which are necessary to justice.

It appears that a certain man may be unable to look after his own affairs; or a will has been written; or a person is behaving in an abnormal enough fashion to raise the question of his committability. These matters are *vital* to the person concerned and to the State, and only persons trained in the medical sciences and especially in psychology and psychiatry can speak with such authority as serves justice.

A crime has been committed. Is the person who committed the act in such a mental condition that he may be tried? If he may be tried, is he responsible? If he be responsible, is he punishable? In most communities of the United States these questions are being decided partly on the basis of opinion evidence coming from those not trained in the medical sciences; and in most cases the "alienist," even when selected by the Court, is qualified by friendship and political loyalty rather than by training and experience as a psychiatrist. This medical aid is far short of the type which tends toward equity and towards justice.

### EQUIPPING THE MEDICAL MAN FOR RELATIONSHIP TO PROFESSION, SOCIETY, AND LAW

The medical man on commencing practice finds himself more or less well prepared to cover those portions of his practice concerning the relationship "physician—individual patient." Soon, however, he is faced with problems concerning his duties and responsibilities under the Law; the matter of privileged communications; narcotic and alcohol legislation; malpractice allegations; and the relation of medicine to one or another type of insurance. He is called to Court as a "fact" or "opinion" witness. He is asked for opinion in matters where pathology or toxicology touch the Law. He will have to obtain permission to make postmortem examinations and know the penalities for performing them without proper authority.

The practicing physician is called on for opinion concerning disease or disability in relation to occupation; and concerning injuries in which there is alleged industrial or other liability.

Questions come to the physician which relate to marriage, divorce, perversion, rape, abortion, impotence, and sterility.

Lastly, and equally important with all of these problems, come those related to the practicing physician's status as a citizen and as a member of a profession and a guild. He must guide his actions so that his conduct may be courteous and ethical in relation to his patient and his brother practitioner.

In most communities of the United States the graduating medical man commences practice without any adequate preparation to meet these problems.

### PRESENT STATUS IN UNITED STATES AND EUROPE

The medical schools of the United States, with a very few exceptions, have made little or no provision for teaching or for service concerning the application of the authoritative knowledge of Medicine and its allied sciences to social problems and the administration of Law. American medical schools should have departments or institutes of Legal and Sociological Medicine adequately equipped and staffed to place Medicine in the position of responsibility, which only Medicine can assume, concerning the medical aspects of an ordered society, and the administration of law. In Austria, Germany, Czechoslovakia, Poland, Italy, France, Portugal, Roumania, Scotland, Sweden and Switzerland we find such institutions existing in full functioning capacity. They are serving society and the courts either by loose, informal arrangement, or by legislative enactment. Departments of research are adding scientific data to our understanding in this field of activity. Instruction is being provided for medical and legal students and for graduates in Law and Medicine.

### PLAN TO DEVELOP INSTITUTE

At least one Class A medical school in each state should establish such an institute. It would seem logical that the State University, if there be one, rather than some other school, might establish such a department, for a State university is already more intimately related to the State structure. It might proceed at once with the educational end of a program; and might more easily by legislative enact-

ment, or some other procedure, prepare to give its services to the State wherever Medicine touches sociological problems include more definitely pathology, toxicology, laboratory medicine, neuropsychiatry, insurance medicine and traumatology.

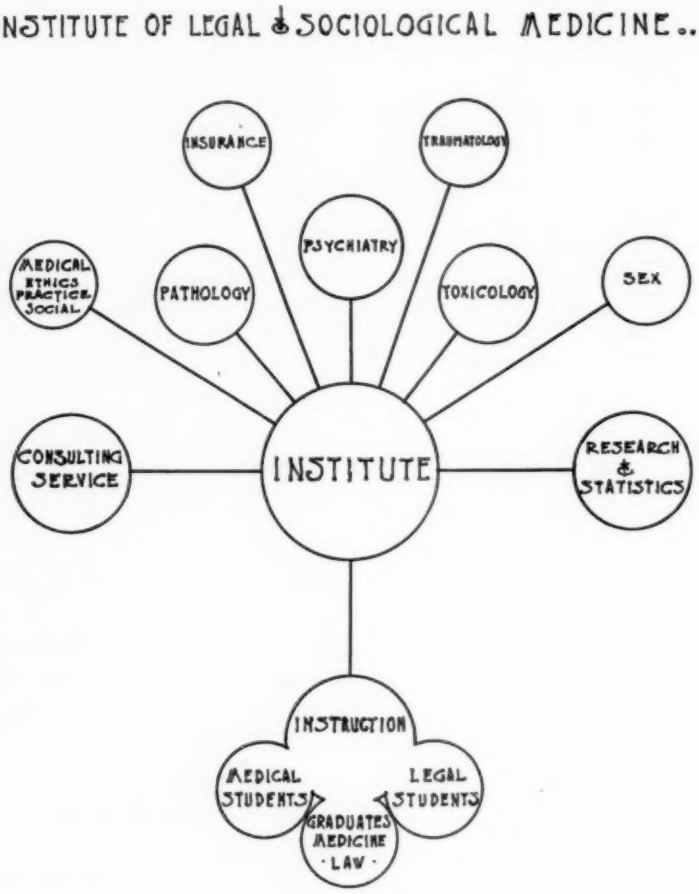


FIG 2

or problems concerned with the administration of law and justice.

It is not necessary to enter at once into the development of a completed plan with a large staff, including paid teachers and employees and a building to house the institute and involving elaborate financing. However the skeleton of an Institute of Legal and Sociological Medicine might be developed forthwith in any class A medical school.

It becomes immediately apparent that both for the teaching and service purposes of such an institute many of the departments of a medical school would necessarily cooperate. The legal aspects of Medicine But when it is stated that these various departments must necessarily be involved, it is not to be inferred that they must undertake at this stage any new and time-consuming duties. There is however a minimum though adequate degree of co-operation and correlation that must be arranged, if the Institute shall be what is planned. The Institute's policies and growth should be determined by interdepartmental conferences, rather than by any single individual or by the department itself.

Legal and sociological Medicine necessarily runs through and involves various departments. Accordingly, it is contemplated

that the heads of the departments of Pathology, Pharmacology and Neuropsychiatry should become consultant and advising directors to the Institute. These individuals should sit in conference with the Director of the Institute and lay out the work which shall be done at this time; and plan the future development of the Institute. There will be need for further conferences at suitable intervals. In addition to these periodic conferences of heads of departments, each department chief would be called upon to give one or two lectures of a required course of 16 lectures to undergraduate fourth-year medical students.

### PHASES OF DEVELOPMENT OF THE INSTITUTE

Phase 1. There should be arranged immediately a course of 16 lectures for undergraduate fourth-year medical students, such course to cover as completely as practicable, in such a number of lectures, the various aspects of Legal Medicine and Medical Sociology.

Phase 2. It is contemplated that in due time the following courses should be arranged:

(a) A course for graduate medical students
(b) A course for undergraduate legal students
(c) A course for graduates in law
(d) Special intensive courses in one or another aspect of the field.

Phase 3. It is contemplated later to develop a demonstration unit in each subdepartment, for example, in Toxicology, Pathology and Neuropsychiatry.

A demonstration of accurate and complete toxicological studies and reports in a series of legal cases where poisoning has been the central factor.

A demonstration of accurate and complete pathological studies and reports in a series of coroner's cases or other legal cases where post-mortem studies have been important elements.

A demonstration of accurate and complete case studies, including medical, neurological, psychiatric, psychological and sociological examinations in a series of juveniles and adults charged with crime, such a study to be made in a clinic developed in connection with the local courts and prisons.

A demonstration of accurate and complete case studies in problem children and children with problems, who have not contacted the courts because of delinquency. This demonstration will be similar to that of the various child guidance clinics now functioning.

It is understood that a medical school may be unable to reach out and do a big job in any of these directions before a considerable time has elapsed and before an adequate financing program has been arranged. It appears, however, that the skeleton of each of these units and the working plans for their function should be available at the Institute, and that wherever possible, a small completed demonstration of the respective type of work in a given field should be available. These matters are a part of the program of the Institute, because there is nowhere today that the public or the State may turn for authoritative information and guidance in these matters. With the Institute established, it would have the responsibility of serving the public in relation to these things.

Phase 4. It is contemplated as time goes on to complete the development of subdepartmental units of Pathology, Toxicology, and Psychiatry; and later of other subdepartments. At the present time these subdepartments would be represented in the lectures provided for undergraduate students.

Phase 5. It is contemplated that eventually the Institute might find itself completely financed, adequately staffed, housed in its own buildings, and fulfilling all of the purposes heretofore and hereafter discussed.

*Phase 1 of Development of Institute.* This phase of the development of the Institute is available for immediate adop-

tion for any medical school. It requires, first, the establishment (a) of a new course of lectures; or (b) the modification of the existing course in legal medicine, if there be one; or (c) some arrangement whereby most of the field is covered in the course in legal medicine proper, and with certainty it is determined that the other lectures are specifically covered in other courses.

Secondly, to put this phase of development in operation, the sixteen lectures should be given *only by authorities in the respective fields*. This is a most important feature of the plan. Heretofore in most medical schools the course in legal medicine has been a sort of "step sister." The lectures are usually given by one individual. Often he is some young physician who has plenty of time on his hands and little or no experience in any field of medicine; the same type of physician who is often selected for lectures to nurses. In other schools we find some young attorney giving the entire course. It may well be that in certain of purely legal matters concerned he is properly qualified. Obviously he lacks adequate qualifications to deal thoroughly with toxicological, pathological, and psychiatric problems with legal implications. In still other schools we find a retired physician, a dean, a gynecologist, psychiatrist, or some other medical man giving the entire course. No one person may speak with authority in all of the fields covered; and there are immediately available in the faculties of medical schools, persons who may speak with authority in the respective fields to be covered.

Accordingly, first of all a staff should be set up for the Institute. The following is suggested:

STAFF OF THE INSTITUTE

*Director of the Institute*

The Professor of_____
(Preferably Professor of Psychiatry or Pathology.)

*Consulting Directors*

The Professor of Pathology
The Professor of Pharmacology
The Professor of Neuropsychiatry.

*Teaching Staff*

The Professor of Law
The Professor of Psychiatry
The Professor of Medicine
The Professor of Pathology
The Professor of Pharmacology
The Professor of Surgery
The Professor of Gynecology and Obstetrics
A Superior Court Judge
The Coroner of the County
The Medical Director of the Industrial Accident Commission*
Attorneys at Law, Doctors of Medicine } Lecturers in Legal Medicine.

There are listed below the required lectures, subjects to be covered in each, and by whom the lecture is to be given.

REQUIRED COURSE OF LECTURES FOR UNDERGRADUATE MEDICAL STUDENTS

A. *Concerning the legal aspects of Medicine, and the medical aspects of Law.*

1. Introductory. Outlining course: Considering briefly each of the subjects to be covered. Introducing attorney who will give a "Primer of Law" as it relates to the practicing physician; defining legal terms; skeleton of legal procedure.
—Director of the Institute and an Attorney of Law.†
2. Certain legal aspects of medical practice. Dealing specifically with medical practice acts, licensure, rights of

* Or its equivalent.

† In the School of Medicine of Stanford University this course is under the direction of the author, an Associate Professor of Medicine, Psychiatry. "An Attorney at Law" indicates much more than the title. It is suggested that each medical school do as has been done at Stanford in the selection of this attorney. There were three outstanding attorneys in California who might speak with authority in this field. They were those who had served as counsel for the California Medical Association over a long period of years. One of these men who had twelve years of such experience and therefore had handled large numbers of alleged malpractice cases and every other sort of legal implication of medical practice, was selected to give the lectures assigned to "Attorney at Law."

physicians, obligations of physicians, vital statistics, alcohol and narcotic laws, and malpractice.

—An Attorney at Law, Lecturer in Legal Medicine.

3. The Physician in Court as a fact witness, and as an opinion witness.

—A Staff member with adequate court experience.*

B. *Concerning the legal aspects of Pathology, Toxicology, and Laboratory Medicine.*

4. Manner of determining, and legal importance of death from cold, heat, burning, starvation, injuries, lightning, electricity; various types of wounds; suicide versus homocide; etc.

—The Professor of Pathology.

5. Signs of death. Identification of living or dead. Permission for autopsies; liability for unpermitted autopsies; methods of autopsy; records; the Coroner's office.

—The Professor of Pathology.

6. Toxicology. Its medicolegal relations.

—The Professor of Pharmacology and Toxicology.†

C. *Medicolegal aspects of Insurance.*

7. Medicolegal aspects of life insurance, industrial accident insurance, health insurance, personal liability insurance, and so-called malpractice insurance.

—An Attorney at Law. Lecturer in Legal Medicine.

D. *Medicolegal aspects of accidents.*

8. Traumatology. Effects of external violence on body structures and body function; examinations, reports, estimates of prognosis and extent of disability; relations to insurance, to litigation, etc.

—The Professor of Surgery.‡

E. *Medicolegal aspects of sex.*

9. Marriage and divorce. Perversion. Rape.

—The Professor of Psychiatry.

10. Abortion, birth and illegitimacy. Infanticide. Impotency and sterility.

—The Professor of Gynecology and Obstetrics.

F. *Legal aspects of Psychiatry.*

11. General relationships of mental disorder to Law. Medical "Psychosis" and "Legal Insanity."

—Professor of Psychiatry.

12. Psychiatry and Civil Law. Wills, competency, care of mentally ill, commitment of mentally ill, etc.

—Professor of Psychiatry.

13. Psychiatry and Criminal Law. Triability, responsibility, punishability, etc.

—Professor of Psychiatry.

14. Psychoneuroses and malingering in injury cases.

—Professor of Psychiatry.

G. *Medical Ethics and Social Medicine.*

15. Medical Ethics. The physician's responsibility in social movements with medical phases. The physician's duties as practitioner and citizen.

—A qualified Physician.

16. Laws and Legislative trends in relation to Social Medicine.

—A qualified Physician or Attorney.

### ELECTIVE EXTRAMURAL LECTURES AND DEMONSTRATIONS

It is almost the universal experience of medical men to have their first taste of Commission or Court, or any form of legal machinery, when they find themselves summoned, like a bolt out of the sky, into a strange arena for which they are unprepared and in which they find their expected embarrassment warranted or unwarranted. Certainly an earlier visit to the "law in action" at a time when one is an onlooker rather than one involved, is in order.

Accordingly, coincident with the lecture course being given to undergraduates there should be arranged the following extramural demonstrations and lectures; pre-

* A staff member with adequate court experience. The Director of the Course gives this lecture, drawing on an experience of appearances in court in over 350 public liability cases: some 50 homocide cases: and large numbers of will contests, competency hearings, commitment proceedings, etc.

† The Professor of Pharmacology gives this lecture as a part of his own courses in Pharmacology.

‡ This lecture is given by a member of the Surgical Staff with a large industrial accident experience.

scribed if possible; but in any event elective.

(1) A visit should be made to an Industrial Accident Commission at which time the Medical Director of the Commission will discuss the workings of his Department; the Rating bureau will indicate the manner of making estimates of permanent disability; and the students will sit in on a part of an Industrial Accident hearing.

(2) A visit should be made to a Superior Court where expert testimony is being given in a Civil case.

(3) A visit should be made to a Superior Court where expert testimony is being given in a criminal case.

(4) A visit should be made to a Superior Court where a malpractice case is on trial.*

(5) A visit should be made to the Coroner's Office where the Coroner will discuss the procedure of Coroner's autopsies and inquest.

(6) A visit should be made to the Detention Hospital and the Psychopathic Hospital (or equivalent) where the students might learn the manner of entering patients into Psychopathic Wards or State Hospitals; the nature of commitment proceedings, etc.

### FIFTH YEAR OR INTERNE YEAR SERVICE IN INSTITUTE

As time goes on it may become possible to permit students to spend the fifth year in studies and in research work in one or another subdepartment of the Institute, in lieu of interne work in some other department of the Hospital and Medical School. This training will develop scientifically trained physicians to fill the roles of legal toxicologist or pathologist, alienist, criminologist, etc.

### POSTGRADUATE COURSES

Outlines of courses for graduate medical students; for undergraduate legal students; for graduates in Law; and special intensive courses in one or another aspect of the field should be developed after the Institute has been established. The matter of complete courses of training leading toward appropriate degrees should be considered.

### THE INSTITUTE'S RELATIONS

The Institute should be administered through the Director, he being responsible to the Dean and Faculty of the Medical School. The Professors of Pathology, Pharmacology and Neuropsychiatry should serve as Consulting and Advisory Directors.

Appropriate working relations should be made as between the Institute and Departments of Pathology, Pharmacology and Neuropsychiatry and other departments of the Medical School.

The Institute should be charged with the responsibility of keeping in touch with, and establishing working relationships with the courts and appropriate state, county, and municipal institutions, including penal institution, mental institutions, courts, district attorneys, probation and parole departments, the coroner, Industrial Accident Commission, immigration authorities; etc.

The Institute should have the responsibility of keeping in touch with, and establishing working relationships with appropriate state departments; e.g. (in California), the Department of Penology, Social Welfare, and Institutions.

The Institute should keep in touch with the work of, and establish relationships with appropriate social bodies, including the State Medical Association; the State Bar Association; Community Chest; etc.

In short, the Institute should, on behalf of the Medical School, act as a fact finder in relation to, and carry out the Medical School's policies in relation to, those social and public groups or bodies which are concerned with the medical aspects of social adjustment and the administration of law and justice. (See Fig. 1 for dia-

* Following these visits to Court, the judge who is a member of the teaching staff should have a symposium with the students to discuss the medicolegal aspects of the cases.

grammatic presentation of Institute's relationships.)

### THE INSTITUTES FUNCTIONS

*Instruction.* The Institute should, as already discussed, provide at this time a required course of 16 lectures for undergraduate fourth-year medical students; and an elective extramural course of lectures and demonstrations.

The Institute should prepare courses for undergraduates in Law; and for graduates in Medicine and Law; and special intensive courses in various aspects of the field of Legal and Sociological Medicine.

*Subdepartments.* The Institute should plan and develop subdepartments in Pathology, Psychiatry, and Pharmacology as soon as practicable, and later subdepartments of Medical Ethics and Practice, Insurance, Traumatology, etc.

*Consulting Service.* The Institute should develop a library and archives and should preserve case records and specimens and exhibits and develop plans and demonstrations of units of work in its various departments. These would be available, together with the personal services of the staff, for opinions and advice in those matters which relate to the medical questions involved in the orderly running of society and the administration of law and justice.

*Research and Statistics.* The Institute should keep careful records of all of its investigations and should undertake research in various matters in the field in which it works and should keep careful check on its activities by statistical and other methods to the end that the Institute might add to the total of knowledge and understanding of the medical aspects of Sociology and Law. (See Fig. 2 for diagrammatic presentation of the Institute's functions.)

### QUARTERS, LIBRARY, ARCHIVES AND MUSEUM

*Quarters.* There should be provided, at once, a single room in the medical school group of buildings, which would be assigned to the sole use of the Institute. The room would serve for consultations and conferences and would house the library and archives and the beginning of the collection of exhibits.

*Library and Archives.* The initial library should include standard and authoritative textbooks dealing with the legal aspects of Medicine and allied sciences. Records of problem studies, records of case studies, skeleton plans of development for subdepartment units, and the completely written-up lectures given in courses should be filed as the beginning of the archives of the department.

*Museum.* Exhibits and specimens should be preserved. Pathological specimens might be preserved, at the beginning, in the Department of Pathology.

### CONCLUSION

There has been presented a program for the development of Institutes of Legal and Sociological Medicine in connection with Class A medical schools.

The program is practical because, among other things, it offers five developmental phases so that a choice may be made as to just how far the plan may be executed at any particular time.

It is strongly urged that Phase 1 be put into effect in every appropriate medical school. It will be found by any school, as it was found at Stanford, that no significant obstacles stand in the way of doing this minimum job.

The author looks forward to the day when one American medical school after another may execute the entire program. Then there will be available in each state or suitable area, an institute to which any person, or social or governmental body may turn for the application of expert medical knowledge to the needs of the social order, law and justice. These institutes will serve to apply the organized authoritative knowledge of Medicine and allied sciences to social problems and to the administration of Law.

# ASPHYXIATION—A BASIC PROBLEM IN MEDICAL EDUCATION*

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NEW YORK CITY

THE problem of asphyxiation as it impinges upon the field of medical education proposes two questions for our consideration. First: Why does the problem of asphyxiation deserve special attention? Secondly: How may the subject be approached from the point of view of the medical school and the hospital interested in the training of internes?

In answering the first question: Why does the problem of asphyxiation deserve special attention?—another immediately suggests itself, and that is: Why should asphyxiation, an ever present problem in medicine, be suddenly thrust forward as a matter of major medical importance? With these questions in mind, it will not be amiss to devote a few moments to the recent history of the present movement designed to prevent asphyxial death.

It is a curious fact in medical history that significant changes frequently have their inception in nothing more important than an altered viewpoint. The present movement to prevent death from asphyxiation illustrates this fact. It has been common knowledge in medical and public health circles that many lives are lost each year by drowning, by carbon monoxide poisoning, by asphyxia of the newborn, by electric shock, by accidents of anesthesia, by drug poisoning, etc. The conviction, however, that these fatalities are practically all due to the immediate effects of oxygen deprivation, that is, asphyxiation, is by no means so generally understood. While the first treatment indicated and practiced is artificial respiration, even this fact did not serve to unite these diverse conditions under a generic head, that of asphyxiation, until the latter part of 1932. At this time, a physician whose work was limited to the care of the unconscious and who possessed a flair for originality, conceived the idea that many conditions causing accidental death were all united by the same common denominator of treatment. This treatment was artificial respiration. If artificial respiration were promptly and efficiently applied, the patient lived; if this treatment was not instituted or was unsuccessfully performed, the patient died, death in each case being due to unrelieved anoxemia or asphyxiation.

Further curiosity resulted in an examination of the vital statistics of New York City which revealed the astonishing fact that the many conditions bringing about or predisposing to asphyxial death totaled more than twice the deaths reported as due to automobile accidents. In view of the fact that the total asphyxial deaths due to these causes were, of necessity, deduced from the available records, nevertheless the conservatism of these deductions suggesting an annual totality of 50,000 deaths in the United States, contained so much truth that asphyxia as a major medical problem is everywhere finding acceptance.

The essence of this acceptance is to be found in the acknowledgment that the present form of returning death certificates must be altered. Dr. T. F. Murphy, Chief Statistician for Vital Statistics, Bureau of the Census, Washington, D.C., says on this point:

> The lack of authentic data relative to asphyxial deaths will require much more complete information on the standard certificate of death and a much more detailed study

* Read before the Society for the Prevention of Asphyxial Death.

of the subject than is now given in the annual reports on mortality.

The acceptance of the problem of asphyxiation on the part of leaders in administrative and public health circles is unique. It is rare indeed to find a movement officially born in February, which would be permitted to hold a Conference at the New York Academy of Medicine in May, a Conference, by the way, made up of a group of leaders any single one of whom would assure the success of a meeting. Reference to a few of these names will suffice: Dr. Chevalier Jackson, Dr. Haven Emerson, Dr. Yandell Henderson, and Dr. Edmund B. Piper.

The importance of the problem discussed at the May, 1933 Conference was immediately grasped by the Editor of the *Journal of the American Medical Association* who devoted at least sixteen columns of abstract and a special editorial to this movement. Let me quote one paragraph of the editorial that is of special interest:

> The Journal has deprecated repeatedly the formation of special societies for every tiny phase of medical investigation and practice; nevertheless, this new organization may serve an especially useful purpose in meeting a trend in medical practice that cannot be met in any other way.

The importance of the work may also be indicated from the sponsors who have seen fit to associate themselves with this new movement and whose names appear on the literature of the Society.

That the sponsorship of this new idea is not only individual but corporate as well will be seen in a resolution passed by the New York County Medical Society last December from which I will quote the following paragraph:

> . . . that the Medical Society of the County of New York approve the aims and purposes of the Society for the Prevention of Asphyxial Death, and . . . endorse the program of the Society for action looking toward reduction of the death rate from Asphyxiation.

Last but by no means least, is the fact of the present Conference and the gathering of this evening.

The facts which I have stated will, I believe, answer the first question: Why does the problem of asphyxiation deserve special consideration?

We may now consider briefly the policy which may be pursued in presenting this problem to the medical student while at school and while serving as interne. In the final analysis, the solution presents two distinct phases. It must be viewed first of all as a basic problem of medical education. The solution of *this* phase will probably require from ten to fifteen years. Secondly, it is to be viewed as an immediate emergency problem. We cannot disregard a continuous loss of 1900 lives each week in the United States with equanimity. Our general discussion confines itself as it relates to medical education.

As such a basic problem in medical education asphyxiation may be dealt with in the curriculum of the medical school by one of two methods of approach; first, by a policy of segregation in which emphasis is placed on all matter in the departments of anatomy, physiology, therapeutics, thoracic surgery, pathology, electrodynamics and any other department which may have a bearing upon asphyxiation. Secondly, by pursuing a policy of integration in which a new department is organized for the specific study of all matter dealing with asphyxiation.

To elaborate: Assuming that it is found desirable to meet the need by segregation, contact should be made with the heads of the various university departments with a view to bringing before these departmental heads the importance and the size of the problem of asphyxiation. For example, it may be requested of the professor of anatomy that when he lectures upon the muscular and cartilagenous structures of the mouth, the pharynx, the larynx, the trachea and the bronchii, emphasis be placed upon the form of these structures in their relation to the problem of ventila-

tion; that the musculature of the trunk involving the muscles of respiration be studied with a view to their relation to the respiratory function.

The head of the department of physiology would be asked to emphasize the factors of internal and external respiration, the effects of paralysis upon the respiratory muscles regularly occurring in the relaxation of anesthesia and asphyxia. The vital problems of tidal volume, the diffusion of gases, partial pressures and surface area for gas absorption and elimination should be stressed. The importance of respiration in stimulating and supporting circulation should be emphasized in order that the value of mechanical respiration as a support to the failing circulation may be grasped by the student.

Of even greater importance in the department of pathology should be stressed the mechanical effects of abscess, solid tumor, edema and aspirated fluid in the airway. Actual pathological specimens should be assembled to demonstrate the mechanical interference brought about by retropharyngeal abscess, tumors of the tongue and fauces, tumors of the vocal chords, external pressure upon the trachea, pressure and reduction of the lung area by fluid, by atelectasis or by bronchial obstruction. If these conditions are demonstrated in their relation to asphyxial death, subsequent treatment of these conditions by the students will be more successful.

The department of otolaryngology would be required to demonstrate the technique of handling the pathological conditions already referred to. It should demonstrate the simplicity and ease of laryngoscopy in the asphyxiated and in the anesthetized patient, since emergency resuscitation of the completely asphyxiated frequently turns upon this one procedure.

In addition to the foregoing departments which should be asked to contribute to the intensive instruction of the medical student, the department of thoracic surgery offers perhaps one of the most instructive and vital fields. In this department is continually demonstrated the phenomena of pressure as applied both within and without the cavities of the chest.

In addition to the direct references alluded to, instruction in the department of therapeutics as it deals with the vapors of ether, chloroform and ethyl chloride; and the gases: carbon dioxide, oxygen, ethylene, etc., should be utilized.

The department of medicine offers the large field of acute pulmonary complications and cardiac embarrassment resulting in anoxemia.

The foregoing is merely suggestive and will serve to indicate some of the specific matter which may be segregated in various departments and brought to the attention of the student in order to impress him with the significance of the problem of asphyxiation. Such a method of approach immediately suggests the difficulties to be faced. The head of each department must be personally approached and his interest aroused before we can expect him to emphasize the matter which we believe should be stressed.

The second approach to this problem may be made by a process of integration, namely, by assembling under one head all correlated facts. The chief resistance to such a policy is its novelty and the objection that we already have too many departments. Upon reflection, however, it will be conceded that the difficulties to be encountered by segregation are greater and offer much less hope of success.

The problem of the treatment of asphyxiation in the many fields in which it occurs presents one common factor and that is this: in every case, be it carbon monoxide poisoning, submersion, asphyxia neonatorum, anesthetic accidents from both local and general anesthetic agents as well as the treatment of the anoxemias of acute pulmonary diseases and cardiac conditions, gases are used as the major therapeutic measure.

This view, suggested by Yandell Henderson in 1928, is attractive in its logic and

in its practical application. It provides a common ground upon which we may meet and deals with the closely related activities now known as resuscitation, anesthesia and oxygen-therapy. While no question arises as to the desirability of such a terminology in the case of resuscitation, the inclusion of anesthesia in this general group has the appearance of undue and unwarranted novelty. Since by far the larger number of general anesthetics, however, are induced and maintained by the use of gases, the vapors of ether, chloroform and ethyl chloride being regarded as gases in practice,—this terminology is not so unreasonable as it may at first appear. The use of conductive spinal and other local methods as well as the employment of basal anesthetics, does not destroy this assumption any more than the use of cardiac stimulants in resuscitation and the use of special medicaments in pneumonia would rule out such a terminology if applied to resuscitation and oxygen therapy.

When it is considered, however, that the proper and full functioning of the present anesthetic service under its surgical supervision is interfered with in no way, and when it is understood that the internist is as free as he ever was to direct the control of his pneumonia patient, the chief difficulties to this departmentalization are removed.

We may conceive of such a department integrating the facts of resuscitation, anesthesia and oxygen-therapy for teaching purposes and rendering, as it were, a definite position and a permanence to modern methods of life saving and the treatment of disease by the use of gases.

It has been suggested and in practice, one frequently sees the Department of Anesthesia called upon to resuscitate the asphyxiated and to provide technical treatment for the pneumonia. However, asphyxial accidents during anesthesia and postoperative pulmonary complications following anesthesia do not occur with sufficient frequency to provide either interest or experience in the care of these patients. In practice, the hospital personnel and the practicing physician do not call the anesthetist to treat asphyxiation due to causes outside of the immediate field of anesthesia. Had this facility been recognized in the past, the immediate major problem of asphyxiation would not have appeared in the present condition for our consideration. It would long ago have been solved.

We must, it would appear, train our personnel and provide our equipment from the vantage point of a new department organized for the purpose of meeting an actual need in the medical field. The various services of the hospital should be educated to look for relief when faced by asphyxial emergencies, to such a specific department designed and operating for the benefit of all services. When an asphyxial accident occurs upon the ward, a trained physician capable of applying modern methods of resuscitation should always be available. If this duty is assigned as a major appointment instead of as a favor to other departments, as is now the case when an anesthetist is called, the morale of such a service will immediately improve.

Trained personnel for this work is daily functioning in the Department of Anesthesia since the anesthetist is familiar with the care of the unconscious patient and has a substantial groundwork upon which refinement of technique may easily be added. Such a functioning department would immediately lend protection to the now uncovered fields of resuscitation and oxygen therapy. The importance and the dignity of this office would promptly be recognized and authority which is now lacking would be conferred. The director of such a department would be interested to assemble facgs relative to the scientific and technical treatment of resuscitation and the problems of internal medicine as they are now faced in oxygen therapy.

A policy of integration designed to reduce the present mortality from asphyxiation as applied to the curriculum of the

medical school appears to offer the greatest hope of relief.

## CONCLUSION

In conclusion, a brief resume of the facts leading up to the present movement designed to prevent death from asphyxiation, appears to confirm the assumption that asphyxia is a major medical problem.

The solution of this problem involves two phases: first, a basic problem of medical education, and secondly, an immediate emergency which presents a total mortality estimated to be 1000 deaths each week in the United States.

The present discussion relates only to the first phase, that of medical education. Two methods are suggested by which the facts of asphyxiation may be brought home to the medical student and to the interne.

The practical difficulty suggested by a policy of segregating the facts of asphyxia in the various university departments inclines us to favor the establishment of a new department which will integrate the present fields of resuscitation, anesthesia and oxygen therapy. The up-to-date physician administering anesthetics must be qualified not only to induce unconsciousness but also to provide every modern facility for the safe return of consciousness and the care of postoperative pulmonary complications.

This special training and experience will not only become available through the organization of a department expressly designed and operated to be on call for all cases of asphyxiation whether in the obstetrical service, the ambulance, or the out-patient department but would also provide a trained personnel familiar with the field of oxygen therapy.

That such a department is much to be desired will at once be recognized by a single reflection: *whom do you call to resuscitate an asphyxiated patient and to care for the oxygen-therapy administered in your hospital?*

[From Fernelius' Universa Medicina, Geneva, 1679.]

# BOOKSHELF BROWSING

## ASPHYXIA: THE HISTORICAL BACKGROUND*

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ASPHYXIA is almost exactly a transliteration from the Greek asphuxia, the Greek upsilon being replaced as is customary by our English "y." The word which has now come to mean a shutting-off of the breath meant in the original Greek a stoppage of the pulse. It was noted from very early times that any incident which shut off the breathing such as choking from the effort to swallow too large an object, or drowning, or strangling, very soon caused a stoppage of the pulse and of the heart beat.

From very early times asphyxiation was recognized as a comparatively easy way of causing death with no messiness about it and yet with the assurance that there would be no uncomfortable coming to afterwards. Strangling was one of the earliest modes of capital punishment. It is still used for that purpose in one form or another throughout the East. Over a large part of the world hanging continues to be one of the modes of legal execution.

Choking in other ways, sometimes with rather elaborate circumstances, has been practiced. For instance, the garrote still continues to be used in Spain and Portugal as well as in the Latin American countries which used to be their colonies. In spite of the gruesomeness of that procedure and the fact that it is rather bunglingly slow in operation, this process still continues to be used. This form of execution is accomplished by means of an apparatus so arranged as to compress what is popularly called the windpipe, that is, the trachea. The instrument is designed to shut off the breath but it accomplishes much more for the fatality by compressing the large arteries and veins which run to the brain through the neck. Almost needless to say any interference with the direct circulation of the blood to the brain very soon brings about serious consequences, first for consciousness and then for life. The rapid tendency to unconsciousness probably makes this form of asphyxiation for legal execution not quite so cruel as it seems to be. In Spain and Portugal strangulation for punishment purposes is effected by the garrote which mainly consists of an iron collar. The criminal who is to be the victim of the apparatus is placed on a stool with a post or stake behind him to which is affixed an iron collar controlled by a screw passing through the post. This collar is made to clasp the neck

* Read before the Society for the Prevention of Asphyxial Death.

of the victim and is tightened by the action of the screw. This proved to be a hideous mode of punishment, more barbarous than the feelings of people as they grew more refined would stand, so an improvement was made in the apparatus, and as it is now operated the point of the screw is caused to protrude and pierce the spinal cord or marrow at its junction with the brain, thus causing rapid death.

Human beings have always been familiar with the shutting off of respiration by means of choking when an attempt was made to swallow something that was too large but which got beyond the back of the gullet and yet could not be forced downwards. It was caught, moving neither up nor down. In children particularly this was sometimes the cause of death. There was a tradition in old Irish medicine that when something like this happened in a child and it began to get a little bluish in the face this was the signal that there was a dangerous blocking of the respiratory passages. The child should be struck firmly on the back between the shoulder blades or a little below them with the open hand in the hope of causing an eructation that would bring the offending object up. Of course there is very little time to spare under these circumstances and another old Irish tradition suggested that the child should be grasped by one or both feet and held up in the air, head down, and then patted on the back.

Among the Greeks in that golden age of medicine which began with Hippocrates they recognized that various forms of inflammation of the larynx might shut off respiration with the production of asphyxiation. Among these diphtheria was probably the most frequent. The word has a Greek etymology but the term was not invented until about a century ago by the Spaniards or perhaps by Bretonneau. Aretius who lived in the first century mentions diphtheria under the term "ulcera Syriaca," syrian ulcers, as if it were a rather common disease. The next mention of it comes among the Spaniards who called it garotillo, that is the little garrote or choking affection. A series of epidemics of this disease were noted and always the tendency to produce asphyxiation was set down as characteristic of it. We had epidemics of it in this country in the seventeenth century when it was spoken of as the disease of the bladders in the throat, a descriptive term for the false membrane which forms above the mucous membrane and portions of which may be removed and look not unlike the sort of tissue that makes up the bladders of animals.

In connection with diphtheria and other obstructions in the larynx that involve shutting off of respiration, the question of opening the trachea came up very early. Tracheotomy, however, was not performed until the case was absolutely hopeless otherwise, so that it becomes easy to understand why it prolonged life only for a time, but the patient was too far gone to be really benefited by it. At least the patient did not die in the midst of the tossing struggle for breath which was so disturbing for those in attendance on these cases and particularly for parents who had to stand by the bedside, utterly unable to do anything to help their child get its breath. The relief afforded by tracheotomy is very great but the constitutional character of the disease led to the exhaustion of the child and death was likely to ensue.

It is not surprising then that tracheotomy went out of use, though with the revival of surgery at the end of the twelfth and the beginning of the thirteenth centuries it seems to have come back into vogue. There were not many things that these surgeons of Salerno, Bologna and Paris missed, and tracheotomy was one of their important revivals of surgical procedure.

Hippocrates suggested the possibility of putting a tube into the larynx in cases where inflammatory or mechanical conditions caused a tendency to the shutting off of breath. In writing the life of Dr.

Joseph O'Dwyer, the inventor of intubation, some thirty years ago, I quoted Hippocrates as mentioning canalization. He suggested that in inflammatory croup with difficulty of respiration a canula should be carried into the throat so that the air could be drawn into the lungs past the obstruction in the region of the larynx. There seems to be no doubt that this suggestion was put into practice by Greek physicians, but there were many practical difficulties connected with its employment, and so it is not surprising that after a time the practice went out of use and this valuable remedial measure was lost to view.

After all when it is realized that every century during the Middle Ages some reference to anesthesia for surgical purposes can be found and yet it entirely went out during the seventeenth and eighteenth centuries, it is not astonishing that more complicated procedures should have been lost as well.

The next that we hear of the use of the canula or tube for the relief of asphyxiation or difficulty of breathing comes in the discussion among a group of French surgeons during the first quarter of the nineteenth century. Early in the century Desault succeeded in placing a tube in the larynx apparently, restoring respiration and lengthening patients' lives, and at least making them more comfortable for some time, though the progress of their disease might eventually prove fatal. Desault's work attracted attention and a student at the University of Paris writing his graduation thesis suggested the use of a gum elastic tube passed down into the larynx in order to allow the passage of air into the lungs in spasmodic and other obstructive conditions. About 1820 Patissier, another French surgeon, suggested that some such remedy as this or mechanical procedure should be employed for edema of the glottis.

In spite of all this interest in the procedure for intubation or, as the French said, canalization of the larynx, the practice died out. Those who knew anything of what happened in France were inclined to think that the French had found that their theories on the subject would not work out in practice and above all that the larynx would not tolerate the presence of foreign bodies in contact with it. The feeling rather generally was that the spasmodic fit of coughing induced by this would surely bring about the ejection of the tube. It was felt also that there were other handicaps connected with it since the practice encouraged infection of the lungs. This was considered to be the principal reason why the practice was given up.

That seemed to be a closed chapter in medicine and surgery and it came to be the feeling that all that was possible had been tried out and that any further investigation or experimentation along those lines was just a waste of time. Almost needless to say there were a great many cases of asphyxiation and diphtheria had a mortality of about 40 per cent, and a great many of the patients and especially the very young children died from suffocation and tossed and moaned and made their progress toward death an awful agony for those in attendance on them, and particularly those who were closely related to them.

Here in New York Dr. Joseph O'Dwyer as attending physician to the Foundling Asylum where they received a great many weakling infants, many of whom had suffered severely from exposure, and practically all of whom as unwanted children were in a low state of resistive vitality, had to witness a number of these nerve-racking deaths from asphyxiation. He resolved to relieve these cases by mechanical means and he devoted some dozen of years to the problem. He tried a wire spring to keep the vocal cords open and then inserted a small bivalve speculum and finally came around to the use of the tubes. It required a great deal of experiment to determine what sort of tube could be inserted without much difficulty and would be retained, but O'Dwyer gradually

worked it out and proceeded to save a great many lives and an immense amount of suffering among the children and the nursing Sisters at the Foundling Asylum who had found the deaths of these infants so hard to bear.

O'Dwyer then proceeded to read a paper before the New York Academy of Medicine in which he described his success with intubation and announced the saving of the lives of children by this means. The best pediatricians in New York, including Dr. Jacobi, were present at this meeting, and all of them were quite sure that there was some mistake about Dr. O'Dwyer's work and that he was not securing the success that he claimed. They told him that intubation had been tried by the Greeks and given up, and then had been faithfully studied by the French, who found it of no value, or at least of very dubious significance. They did not tell him in so many words that he was trying to deceive them but they were quite sure that at least he was deceiving himself and that what he claimed was quite impossible.

O'Dwyer who was the gentlest of men and the most candid and straightforward had an extremely sensitive soul. After the meeting at the Academy was over at which all the authorities on children's diseases brushed aside his work as utterly out of the question, he went home and went to bed and stayed there for three days. The aspersion on his honesty of purpose fairly broke his heart. He had worked long and faithfully and he knew that he was saving lives and suffering, but his colleagues would not believe him and suggested that he was self-deceived if not trying to deceive others. He realized after some time that the only thing to do was to take these cocksure specialists in children's diseases over to the Foundling Asylum and show them what he was doing over there. Just as soon as they were witnesses of what he was accomplishing no doubt was left in their minds not only of his honesty of purpose but of his absolute success in achieving it. I have spoken with Dr. Jacobi about this subject several times and he was quite straightforward in his expression of regret for what they had done that night at the Academy, but he more than made up for it by his praise of O'Dwyer's work.

New York had had a previous experience of this kind that was quite as interesting and that is connected a little distantly at least with the question of asphyxia, so that it may be considered to occupy a place in the historical background. When Dr. Horace Greene took up as a pioneer the practice of the specialty of affections of the throat and nose here in New York he developed a very interesting technique with regard to direct applications to the larynx. When he told the Academy of Medicine of what he was able to accomplish in this matter his medical auditors proceeded to tell him that they were quite sure that anything like direct applications to the larynx were out of the question because they would set up such a spasm as would lead to very severe coughing and very probably to spasmodic closure of the larynx involving asphyxiation. Dr. Greene assured them that he knew exactly what he was doing and invited them to send a committee over to see just what he was accomplishing. Dr. Greene showed the committee the vocal cords through a laryngoscope and after they were accustomed to that sight he applied remedies to the edges of them. One of the committee is said to have said that hereafter if Dr. Greene should declare that he could polish a man's boots through his larynx he would be ready to believe him.

I would not like to produce the impression that New York physicians are over conservative and are very much disinclined to take up novelties, for after all the members of the profession in New York in these cases only did what the profession has always done, that is, put up a strong reaction against anything that seems novel. After all Harvey himself tells us that he lost half his consultant practice as the result

of the publication of his little book on the circulation of the blood. A great many doctors in London and its environs were quite sure that anyone who declared that the blood went running around the body in the way that Harvey said it would must have something the matter with him. That would seem to have been a good lesson for the London medical profession which it surely would not forget, and yet some 250 years later when Lister went up to London and on the urging of friends applied for admission to the London Surgical Society the president is said to have said, "Admit that charlatan Lister? Never! I shall blackball him myself."

It is not only physicians that are conservative but human beings generally. Our first reaction to anything new that is really novel and important is always, "It isn't so, indeed it can't be so," or even, "It is absurd." Then when we have to admit that it is so we say, "Well, it isn't new," and then when its novelty is proved to us we are prone to say that it does not amount to anything anyway. Every discovery must make its way against opposition. That is the way of humankind.

Dr. Trousseau, the distinguished French clinician about the middle of the nineteenth century, reintroduced tracheotomy, that is the surgical opening of the trachea or windpipe so as to admit air through an artificial opening to the lungs. In many cases of blocking of the rima glottidis this procedure afforded relief, but in diphtheria which was so often epidemic in New York very few recovered. The reason was that diphtheria was intensely fatal, but then the children had little resistive vitality. O'Dwyer himself said when asked what led him to think of intubating the larynx that he had been aroused to experimentation in this direction by the complete failure of tracheotomy for some ten years at the New York Foundling Asylum. Tracheotomy is a serious bloody operation and it is always put off until the condition of the patient is quite alarming, if not hopeless. No wonder then that it seemed only to prolong life for a comparatively short time and the patient died of the progress of the diphtheria. Indeed the tracheotomy wound itself seemed in many cases to afford a fresh nidus for the growth of diphtheria bacilli that further weakened the patient and complicated the case.

As regards the early operations for tracheotomy, Gurlt in that great work of his, the Geschichte der Chirurgie, Volume III, gives a full account of all the authenticated instances. This chapter of history goes back to Aesclepiades in the second century before Christ and the history is continued by Gurlt down to the end of the sixteenth century.

Colonel Garrison whom I consulted with regard to this subject suggests that there is not only the possibility but even the probability that pre-historic man may have slit open the gullet of a child or adult suffocating from croup or diphtheria just as savages in Central Africa have been known to perform abdominal section on occasion. When we realize that men here in America before the coming of Columbus did trephining operations when patients complained of intense and continuous headache, or when their disposition had so changed that they seemed to be possessed by a devil so that their friends felt that the evil spirits would have to be let out of the skull, it is easy to understand that tracheotomy would be a comparatively easy development. Bartholomew, the Englishman, in his capitulum on amentia or madness after suggesting change of scene, entertainment and music for the insane says, "If these measures do not succeed then recourse must be had to surgery." There are many other references of that kind to be found in medical history.

The first successful case of tracheotomy in modern times, which begins the modern history of that important surgical procedure, is the operation performed during the course of diphtheria by Bretonneau on Elizabeth de Puysegur on July 1, 1825. It was only after five failures that he succeeded in opening the trachea and affording

relief. The description of what he did is to be found in his book on diphtheria which is an extremely important landmark in the history of that disease. In French it is known by the title, "Des inflammations speciales du tissu muqueux" (Paris 1826). The account of Andre's case is given in the same volume almost immediately afterwards.

Trousseau was the first Parisian clinician to perform tracheotomy for diphtheria and his operation was done on Gustave de Marcillet on November 23, 1831.*

All this I owe to the kindness of Dr. Garrison who used to be at the Surgeon General's Library and is now at the Welch Memorial Library of Johns Hopkins, enabling all of us to do work in the history of medicine that appears learned and recondite and is only due to Dr. Garrison's patient study and ample resources of information on these subjects.

I shall never forget hearing Dr. Thomas Addis Emmet tell the story of his early years as a physician to the City Hospital on the island. He was the visiting physician though a comparatively young man, and while he was making the rounds one day he found a patient suffering from diphtheria and manifestly about to die of suffocation. Tracheotomy was comparatively little used and a very little tried operation in this country at that time, so the house staff was standing idly by and letting the poor fellow asphyxiate. He seemed almost at his last gasp. Emmet called for a scalpel and the resident went to get it (that was the rather easy-going way they did such things in those days) but the house physician was delayed in finding it. It looked as though the man's every gasp was to be his last, so Emmet took out his pocket knife and proceeded to open the man's trachea. Two neighboring patients who were convalescent had seen the doctor's curtness with the resident and his growing impatience when the scalpel was not brought, and they thought that Dr. Emmet had gone crazy and was cutting the throat of this patient, which he was of course doing but in the most highly rational way. One of them attempted to hold Dr. Emmet, who planted his heel in the stomach of the interferer, kicked him over the chair into the aisle, and told him, I'm afraid, to "get to hell out of there." In a moment the trachea was opened and the patient took a deep, long breath and then gradually became conscious, recovering just as the resident arrived with the scalpel.

We are engaged in preventing asphyxial death just as determinedly as was Dr. Emmet. I am quite confident that a large number of those who find their way into eternity through asphyxia will be given a longer lease of life with us as the result of this organization of the Society for the Prevention of Asphyxial Death. Already much has been accomplished, though we would seem to be only touching the surface of the subject as yet.

*Journal des Connaissances Medicales*, Paris, 1833–34.

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## EDITORIAL

## PRACTITIONERS OF MEDICINE AND THE PUBLIC

THERE seems to be in this country a feeling that we are in the very forefront of things medical and surgical. While this may be more or less true, particularly as regards the technique of operative procedures, nevertheless, there are many weak spots in our general record of achievement in prevention and cure. There are problems in these domains that the laborers in the field must leave largely to scientists and investigators, as in the instance of the cancer situation. There are others in which we must lend our aid not only as medical men, but as citizens, possibly to influence legislation or the enforcement of laws, as in the case of the appalling problem of automobile accidents.

In addition to such problems as those mentioned and many others, there are much simpler ones susceptible of betterment that are strictly ours to improve. In some of these the lines of action are well worked out; it only remains for us to carry them through properly. One outstanding condition of such nature is the common disease—appendicitis, which is well known even to the laity, but which is nevertheless doing great havoc. A most remarkable feature is that the results are getting worse instead of better, in spite of our improved technique, improved anesthesias and after treatment. Is this a case wherein "familiarity breeds contempt?"

Passagno found in a study of 250 acute service cases operated upon in McGlannan's clinic at the Mercy Hospital, Baltimore, that it was necessary to institute drainage in 50 per cent. In a collection of 300 private cases, I found that it was necessary to drain in 40 per cent of the acute ones.

The mortality rate from appendicitis in the United States is said to be about 14 to 15 per 100,000. Calculating on the basis of a population of 110,000,000 people, this would mean a death about every thirty-three minutes from this disease in this country and, according to Guerry, there was a gradual rise in this mortality rate between 1920 and 1925 of over 39 per cent. The mortality figures, of course, do not take into account those who recover with some degree of invalidism as a result of complications.

As we are constantly seeing late cases with varying degrees of peritonitis, it seems urgently necessary that we use all the facilities at our command; lectures, articles, radio talks, newspaper notices, etc., to impress upon the profession at large, and also upon the public, the seriousness of this situation. In spite of all that has been said about this dark picture, there are several fundamental facts that have not been "gotten across" to a large percentage of the profession.

First among these is the danger of giving purgatives in cases of abdominal pain that may turn out to be appendicitis or other inflammatory conditions of the abdomen. This is constantly being done in spite of the fact that as long as thirty years ago, such men as Tiffany and others were preaching "give them castor oil and call the undertaker!"

Secondly, surgery, in many cases, is delayed because there is little or no fever. It must be further emphasized that fever is usually slight in uncomplicated cases and it is not unusual in acutely fulminant gangrenous cases to have little rise of temperature.

A third fact that does not seem to be fully appreciated is that in the majority of cases, acute appendicitis is a quick process, a matter of hours, not days.

It is understood that the family doctor cannot and should not ask for a consultation for every patient with abdominal pain, nor rush the patient to a hospital, but he can withhold purgatives (or narcotics) until he is sure he is not dealing with an inflamed appendix.

We should instruct the public along these lines with gratifying understanding on their part.

Walter D. Wise.

*Baltimore, Md.*

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